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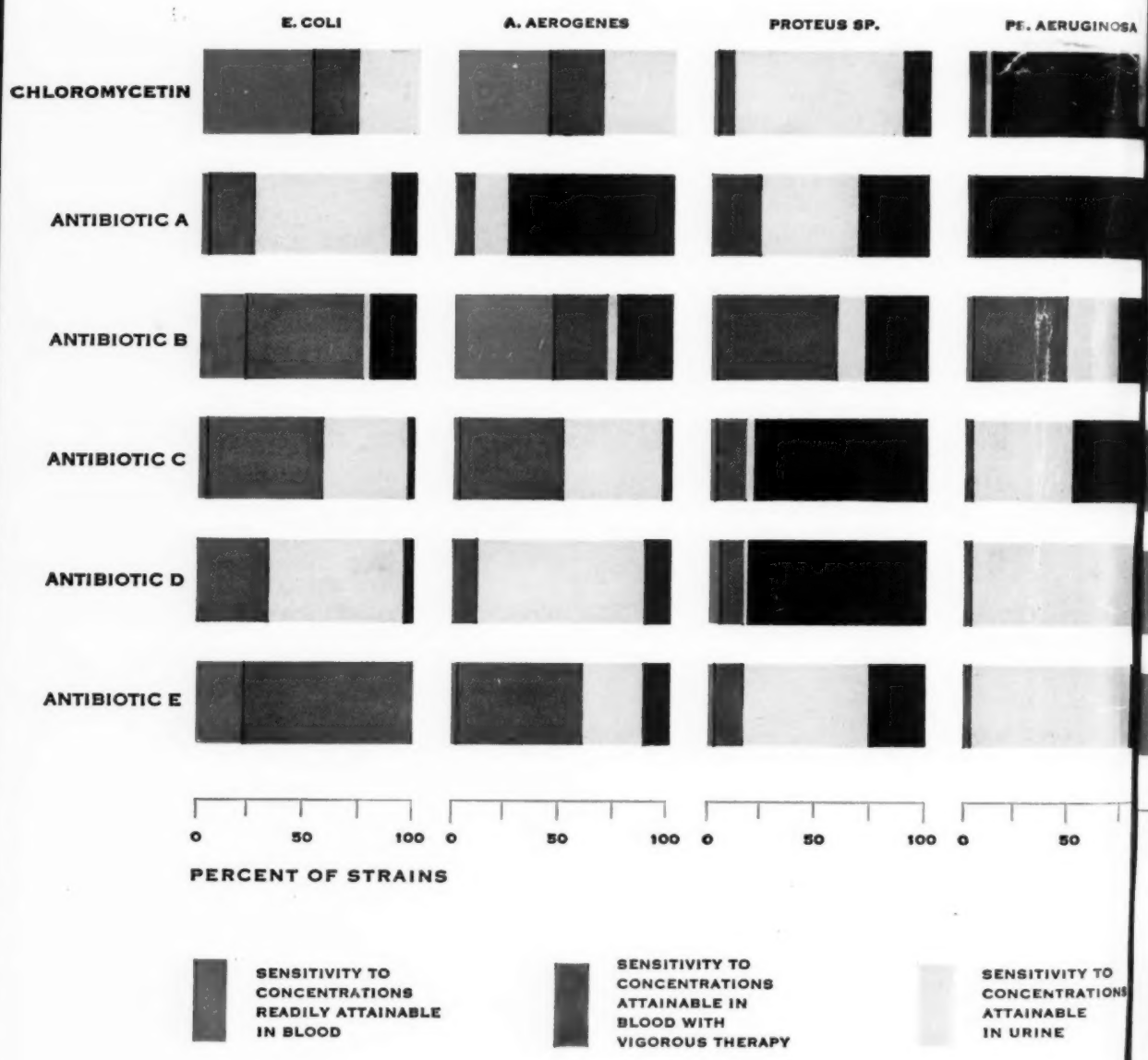
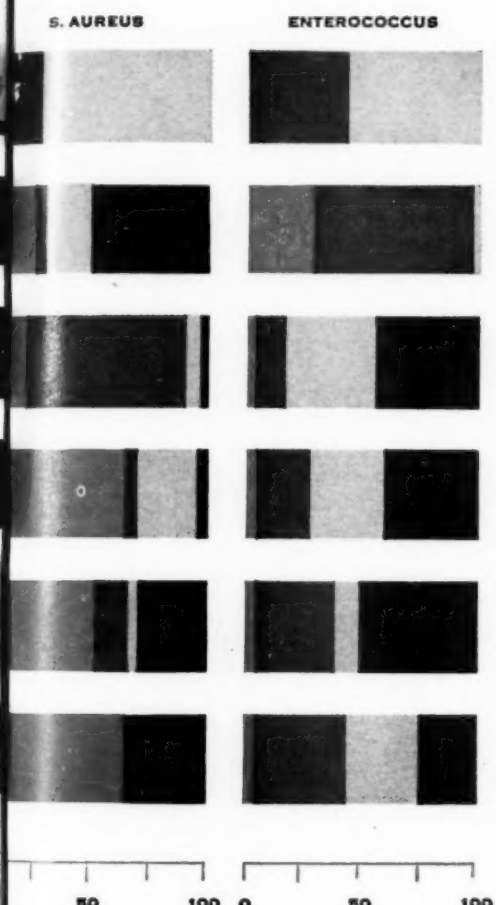
Resistance of Common Urinary Tract Pathogens to CHLOROMYCETIN and Other Major Antibiotics*



less resistance encountered...



Adapted from Kass, E. H.

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References (1) Altemeier, W. A.; Culbertson, W. R.; Sherman, R.; Cole, W.; Elstun, W., & Fultz, C. T.: *J.A.M.A.* **157**:305, 1955. (2) Kutscher, A. H.; Seguin, L.; Lewis, S.; Piro, J. D.; Zegarelli, E. V.; Rankow, R., & Segall, R.: *Antibiotics & Chemother.* 4:1023, 1954. (3) Clapper, W. E.; Wood, D. C., & Burdette, R. I.: *Antibiotics & Chemother.* 4:978, 1954. (4) Sanford, J. P.; Favour, C. B.; Harrison, J. H., & Mao, F. H.: *New England J. Med.* **251**:810, 1954. (5) Balch, H. H.: *Mil. Surgeon* **115**:419, 1954. (6) Sanford, J. P.; Favour, C. B., & Mao, F. H.: *J. Lab. & Clin. Med.* 45:540, 1955. (7) Felshin, G.: *J. Am. M. Women's A.* **10**:51, 1955. (8) Jones, C. P.; Carter, B.; Thomas, W. L., & Creadick, R. N.: *Obst. & Gynec.* 5:365, 1955. (9) Kass, E. H.: *Am. J. Med.* **18**:764, 1955. (10) Stein, M. H., & Gechman, E.: *New England J. Med.* 252:906, 1955. (11) Yow, E. M.: *Postgrad. Med.* **17**:413, 1955.

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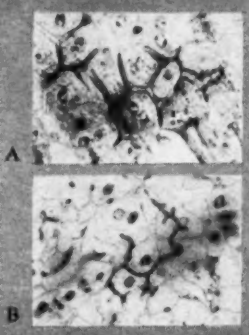
(B) Untreated control.



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(1) Clara, M.: Med. Monatsschr. *7*:356, 1953. (2) Brauer, R. W., and Pessotti, R. L.: Science *115*:142, 1952. (3) Schwimmer, D.; Boyd, L. J., and Rubin, S. H.: Bull. New York M. Coll. *16*:102, 1953.

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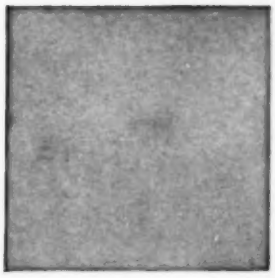
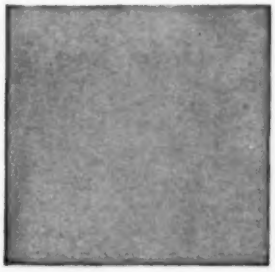
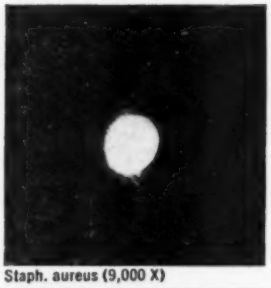


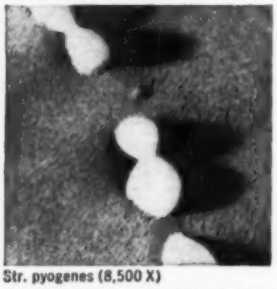
K. pneumoniae (13,000 X)

H. pertussis (7,500 X)

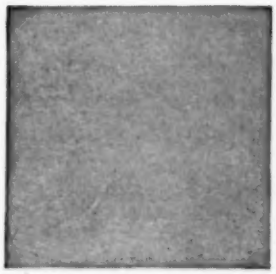




Staph. aureus (9,000 X)



Str. pyogenes (8,500 X)



H. influenzae (16,000 X)

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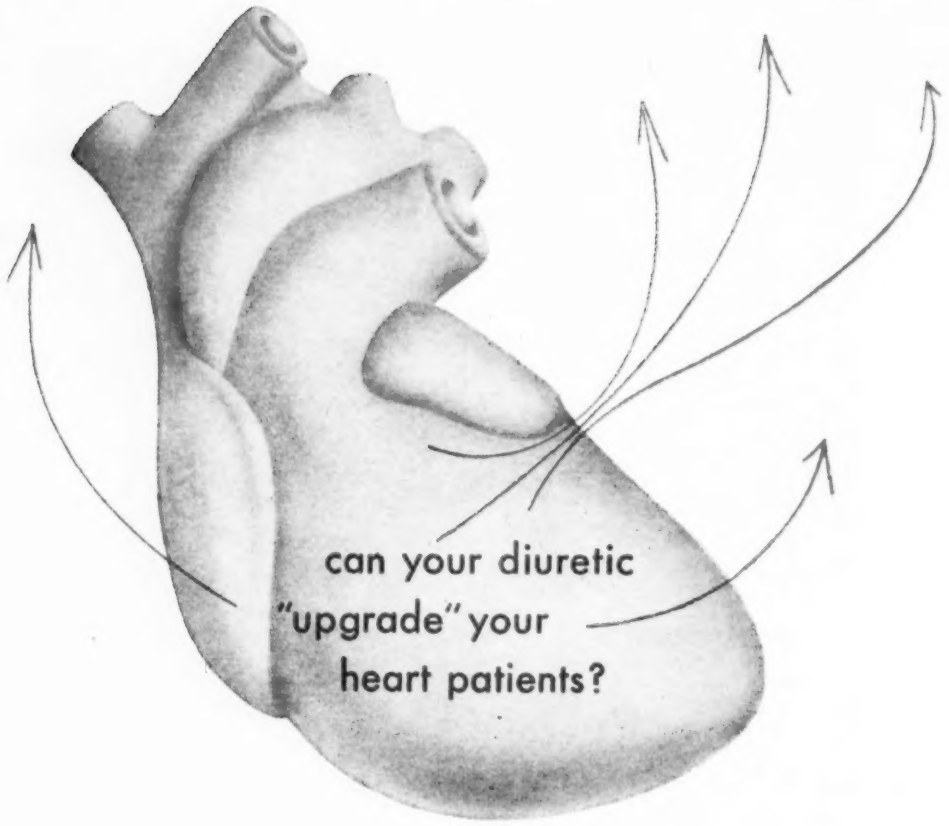

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Terramycin®* has proved effective against an amazing variety of pathogens—ranging from sub-microscopic viruses to large helminthic parasites; findings supported by scientific reports by thousands of physicians on millions of cases. ■Supplied in convenient and palatable oral dosage forms as well as rapidly effective parenteral, topical and ophthalmic preparations.

*BRAND OF OXYTETRACYCLINE

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WHY SENSITIZE

in topical and ophthalmic infections

USE 'POLYSPORIN'®

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POLYMYXIN B—BACITRACIN OINTMENT

to insure broad-spectrum therapy with minimum allergenicity

For topical use: in ½ oz. and 1 oz. tubes.
For ophthalmic use: in ⅛ oz. tubes.

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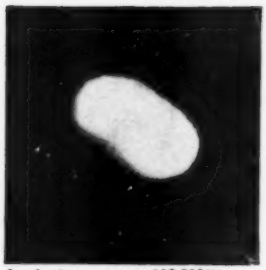
WHEN WRITING ADVERTISERS PLEASE MENTION THIS JOURNAL

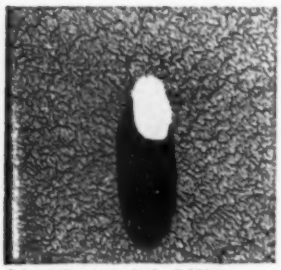
The organisms commonly involved in

Pyelitis

E. coli (8,000X)

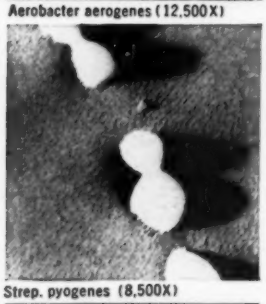


Aerobacter aerogenes (12,500X)



Salmonella paratyphi A (8,000X)

Salmonella paratyphi B (6,500X)



Strep. pyogenes (8,500X)

Strep. faecalis (10,000X)

Strep. viridans (9,000X)

Staph. aureus (9,000X)

Upjohn

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Bronchial asthma
Inflammatory skin conditions

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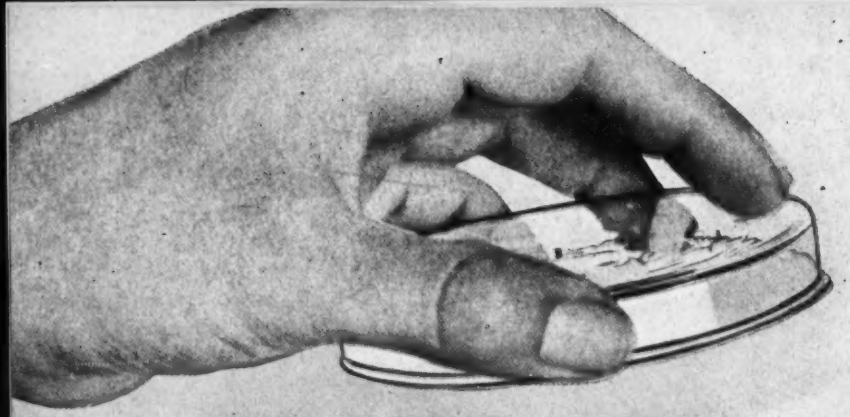
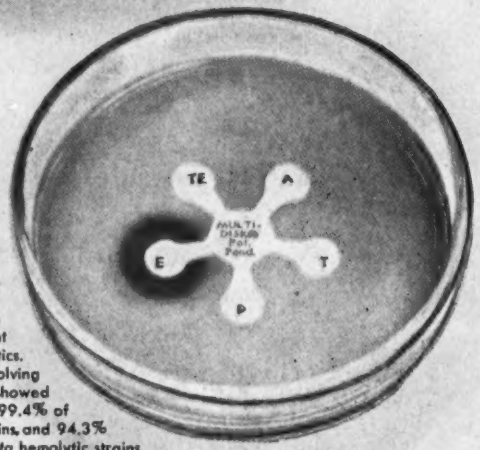
Herein are reported the latest findings on the value of wine as a stimulant to flagging appetite, as an aid to digestion, as a vasodilator, as a daytime and night-time sedative.

We will be glad to send you a copy of "Uses of Wine in Medical Practice" (at no expense, of course). Just write to: Wine Advisory Board, 717 Market Street, San Francisco 3, California.





DESTROYS ENTEROCOCCI

This blood agar plate shows a strain of *beta* hemolytic enterococcus. Note extreme sensitivity of this organism to ERYTHROCIN—yet it easily resists the other antibiotics. Additional data: A study[1] involving 202 enterococcic strains showed sensitivity to erythromycin in 99.4% of *alpha* hemolytic strains, and 94.3% of *beta* hemolytic strains.

specific against coccic infections

Now, you can prescribe *specific therapy* against staph-, strep- or pneumococci by simply writing *Filmtab* ERYTHROCIN Stearate. Since this coccic group causes most bacterial respiratory infections (and since these organisms are the very ones most sensitive to ERYTHROCIN) doesn't it make good sense to prescribe *Filmtab* ERYTHROCIN when the infection is coccic?

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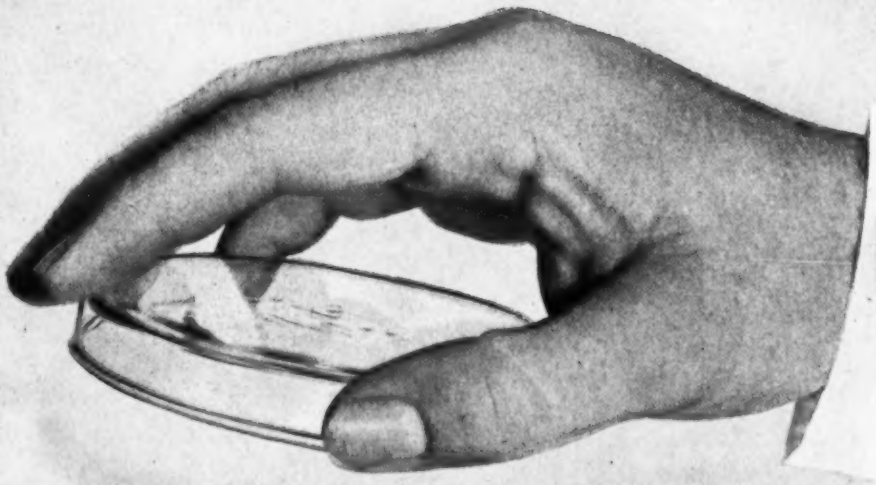

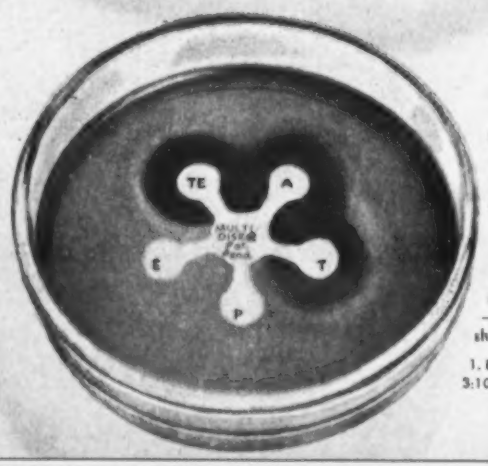

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INTESTINAL FLORA

This sensitivity test shows ERYTHROCIN and the same antibiotics against a typical intestinal strain of *E. coli*. Note that ERYTHROCIN and penicillin do not affect this gram-negative organism —although the other antibiotics show marked inhibitory action.

1. Eisenberg, et al., Antib. & Chemo., 3:1026-1028, Oct., 1953.

with little risk of serious side effects

Since ERYTHROCIN is inactive against gram-negative organisms, it is less likely to alter intestinal flora—with an accompanying low incidence of side effects. Also, your patients seldom get the allergic reactions sometimes seen with penicillin. Or loss of accessory vitamins during ERYTHROCIN therapy. *Filmtab* ERYTHROCIN Stearate (100 and 250 mg.) is supplied in bottles of 25 and 100 at pharmacies everywhere. Abbott

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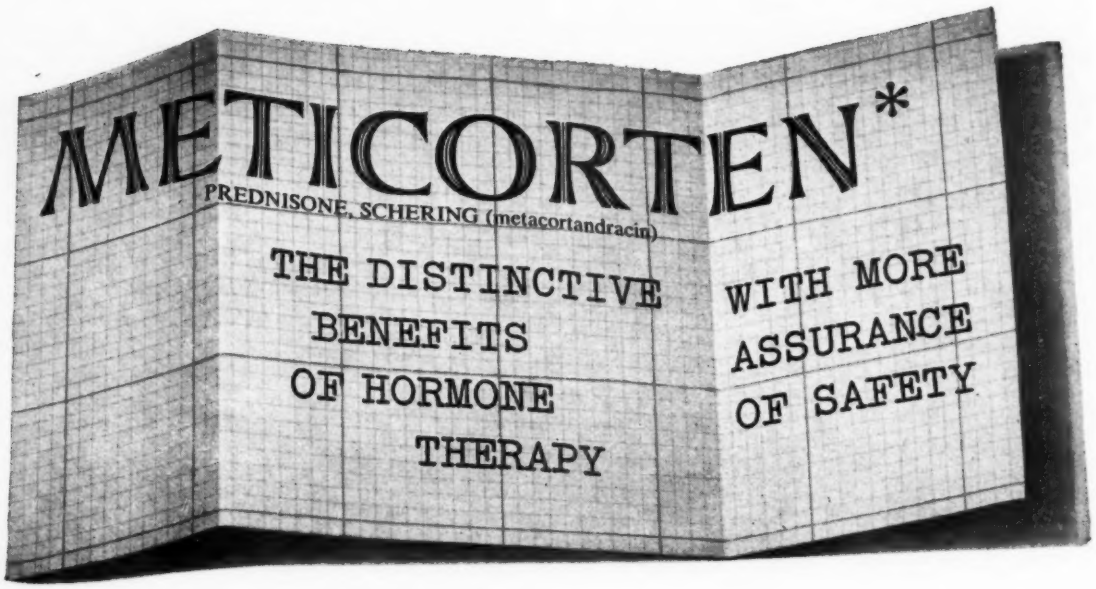

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BIBLIOGRAPHY

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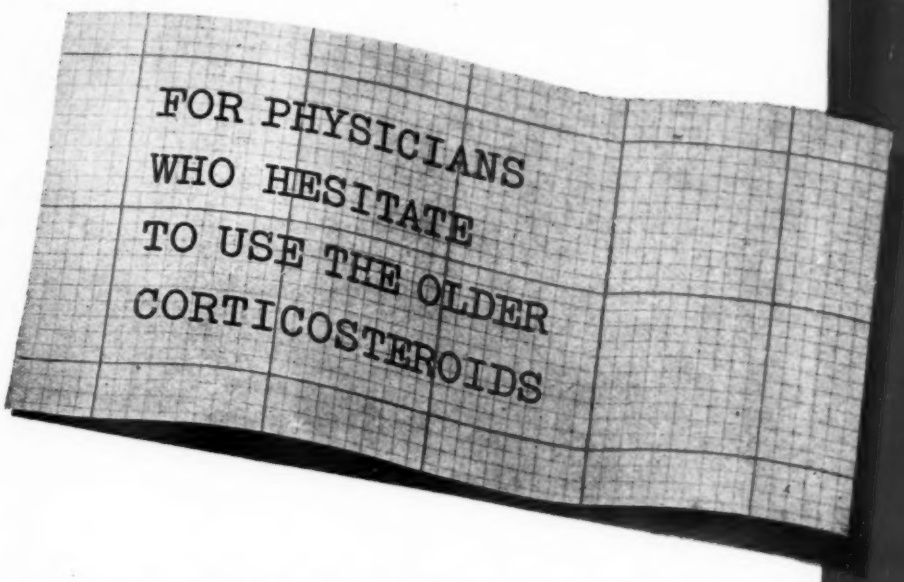
rheumatoid arthritis,

intractable asthma, rheumatic fever, nephrosis, certain skin disorders such as acute disseminated lupus erythematosus, acute pemphigus, extensive atopic dermatitis and other allergic dermatoses, and certain eye disorders



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(1) Payne, R. W.; Shetlar, M. R.; Farr, C. H.; Hellbaum, A. A., and Ishmael, W. K.: J. Lab. & Clin. Med. 45:331, 1955. (2) Bunim, J. J.; Williams, R. R., and Black, R. L.: J. Chron. Dis. 1:168, 1955. (3) Holbrook, W. P.: M. Clin. North America 39:405, 1955.

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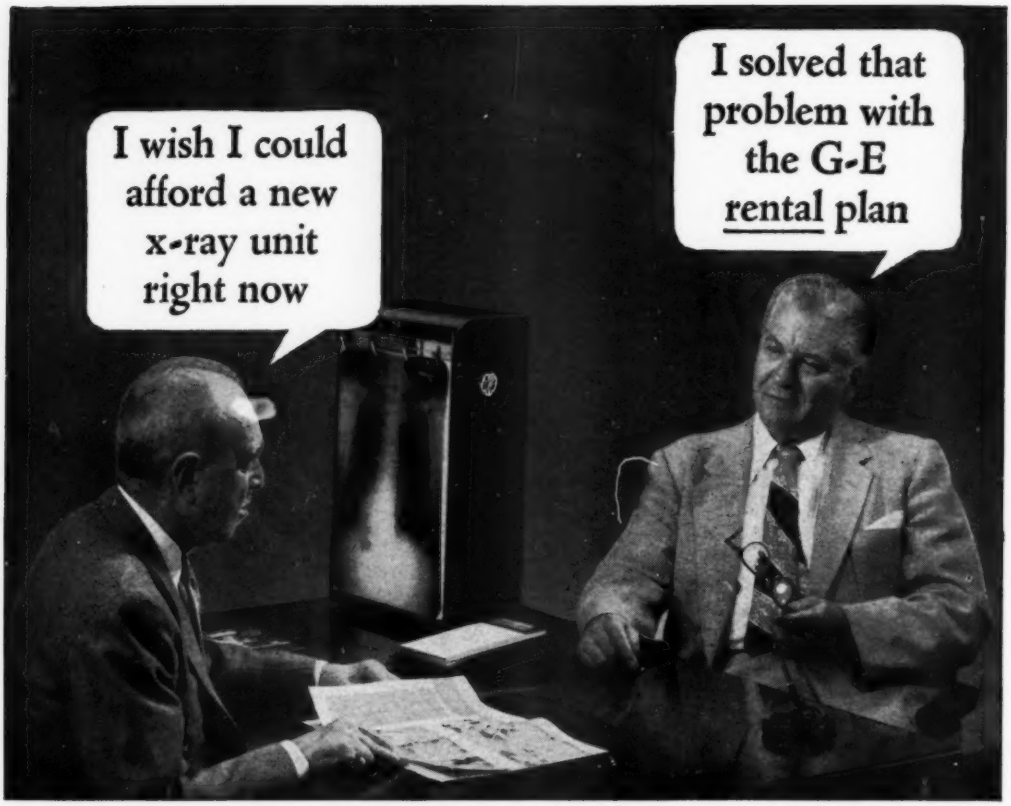

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The organisms commonly involved in

Bronchiectasis

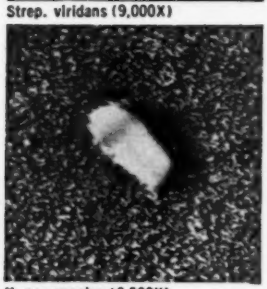
Staph. aureus (9,000X)

Strep. pyogenes (8,500X)

Strep. viridans (9,000X)

Strep. faecalis (10,000X)

E. coli (8,000X)



K. pneumoniae (6,500X)

D. pneumoniae (10,000X)

H. influenzae (16,000X)

Aerobacter aerogenes (12,500X)

Upjohn

ELECTRON MICROGRAPHS

***All* of them are included in the more than 30 organisms susceptible to broad-spectrum**

PANMYCIN*

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Indications:
Rheumatoid arthritis
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"Premarin" —Conjugated Estrogens (equine)

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- Insole extension and wedge at inner corner of heel where support is most needed.
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- Over nine million pairs of men's, women's and children's Foot-so-Port Shoes have been sold.
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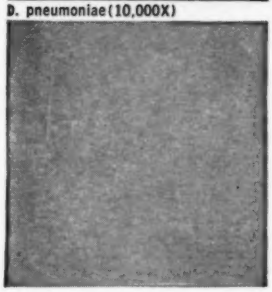
The organisms commonly involved in

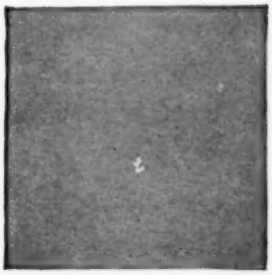
Pneumonia

D. pneumoniae (10,000X)

K. pneumoniae (6,500X)



Strep. pyogenes (8,500X)



Staph. aureus (9,000X)

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Schering's high standards and quality control assure products of unchanging potency and purity for uniform action and clinical efficacy.

minimal cost

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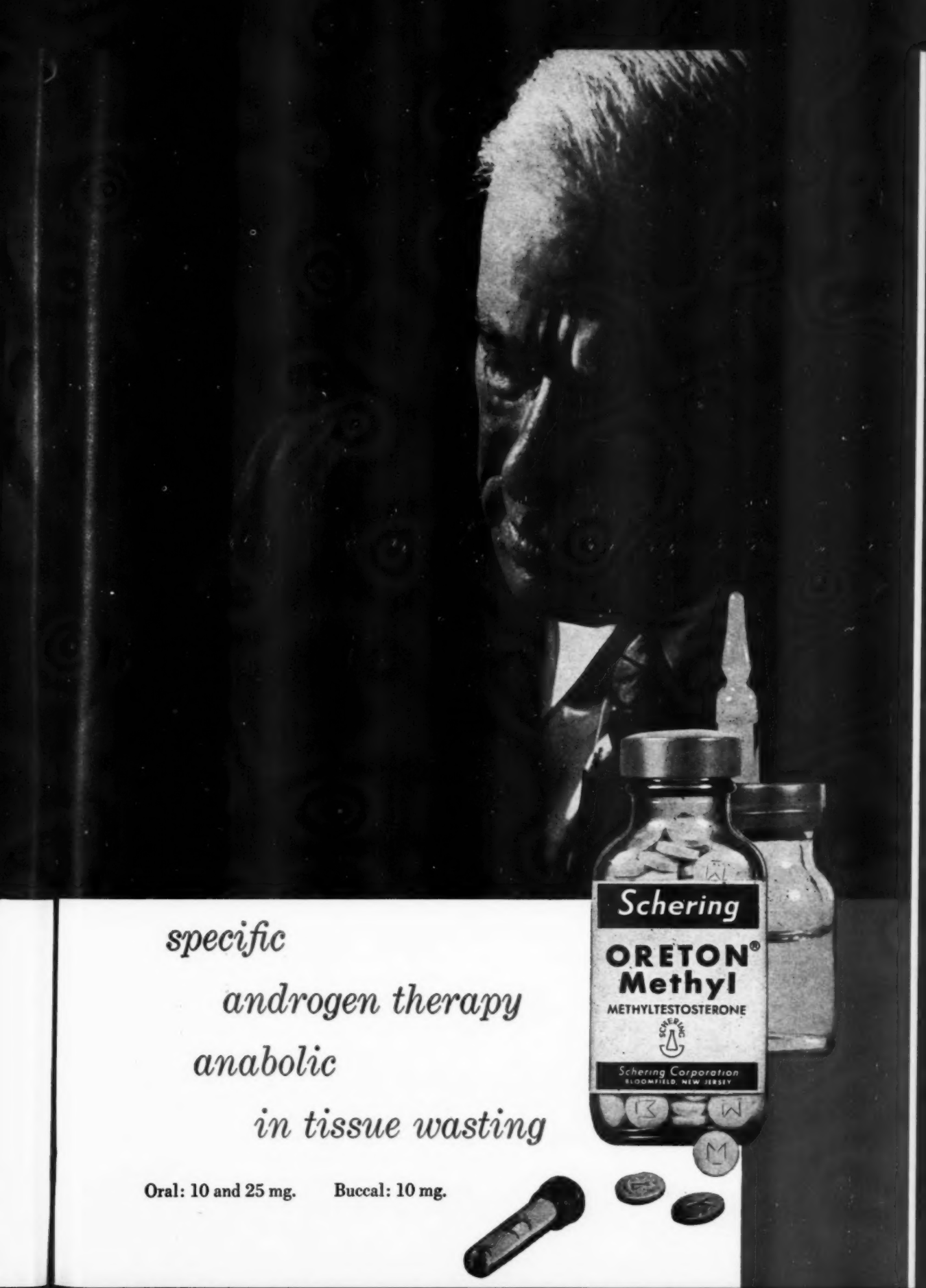

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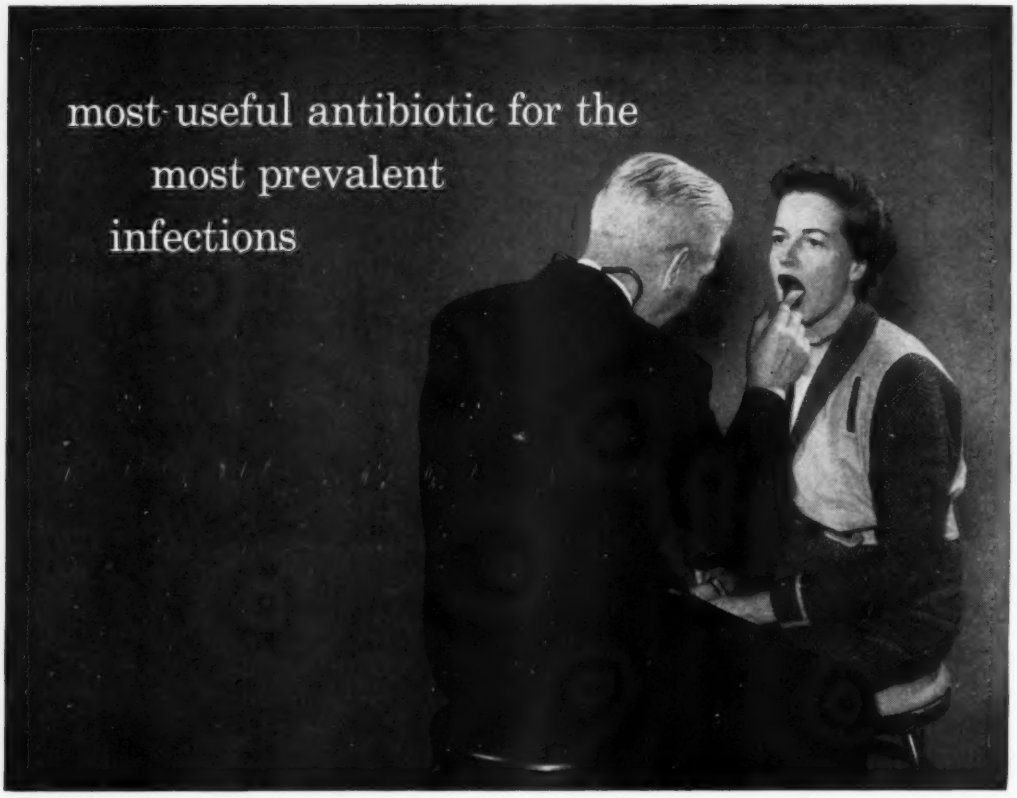
Oral: 10 and 25 mg. **Buccal: 10 mg.**



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'Ilotycin' kills susceptible pathogens of the respiratory tract. Therefore, the response is decisive and quick. Bacterial complications such as otitis media, chronic tonsillitis, and pyelitis are less likely to occur.

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ARIZONA MEDICINE
Journal of Arizona Medical Association

VOL. 12, NO. 11 NOVEMBER, 1955

Original ARTICLES

THE DIAGNOSIS OF HISTOPLASMOSIS AND COCCIDIOIDOMYCOSIS

W. H. Oatway, Jr., M.D.
Altadena, California

Introduction

ONE should be allowed to abbreviate the term histoplasmosis and coccidioidomycosis to 'histo' and 'cox', since the words are so long and clumsy, and since the National Tuberculosis Association has given its seal of approval to the abbreviation 'TB'.

'Histo' and 'cox' are fungus infections which produce disease in a way, and of a type, similar to tuberculosis. The portal of infection, the primary lesions, later lung lesions, disseminated lesions, the x-ray appearance, the delayed skin-test reaction, the antibody formation, and the microscopic pathology are all similar to those of TB . . . Each disease may also occur *with* TB . . . 'Cox' and histo' complicate the study of lung disease, but make it more exciting, and require eternal care and vigilance. We must constantly be medical detectives, and quite often we will have only circumstantial evidence of a crime, since the diagnostic methods are imperfect and the clues do not persist.

The two diseases differ in their *relationship to California* and the Southwest. 'Cox' is an *"export"* disease. It originates here; few other places 'grow' it; and we share its effects with the whole country . . . 'Histo' is an *"import"* disease, since the American source is chiefly in the central United States. What we see in our offices and hospitals is the result of an emigration of its victims from the east, and their appearance is both occasional and surprising.

Read before the California Trudeau Society, Los Angeles, March 18, 1955.

Discussion of Coccodioidomycosis

You know the major endemic areas of 'cox' which include the southern San Joaquin Valley in California, Southern Arizona, Mexico, and west Texas, plus edges of New Mexico, Nevada, and Utah . . . 'Spotty' endemic California areas include a portion north to Merced and Modesto, west to Santa Barbara and Ventura, and parts of San Diego, San Bernardino, Riverside, and (recently) Los Angeles counties. Reports indicate that cases have originated closer and closer to the heart of Los Angeles County . . . These are all arid or semi-arid areas.

The causative organism is a fungus, coccidioides immitis. The terminology is prolix and confusing, but in general the fungus may live in two phases. The *vegetative (or saprophytic)* phase occurs in the soil, where the chief form is the chlamydospore. Cultures produce the same hyphal chlamydospores. They are light, blow easily, either in dust or from the culture, and are highly infectious for animals and man thru the respiratory tract . . . Almost all residents of an endemic area become infected, though one infection confers immunity. A few breaths of a windstorm may infect a transient in such country . . . Animals may be vectors and reservoirs, but to an unknown extent. The fungus lives in the soil for years. The parasitic form lives longer than the saprophytic form in soil experiments, a fact which may be of epidemiologic importance.

The *parasitic* phase consists of a change from chlamydospores into sporangia (or spherules), which then develop endospores, mature, rupture,

and repeat the cycle . . . The two phases may persist independently without altering the character of the fungus . . . It should be stressed that the spherules arrive in the soil by expectoration or excretion from the body. They develop hyphae in 1 to 2 days . . . The peak incidence of human infection is in the dusty seasons. Dust control helps cut the incidence. Spherules are said not to be contagious, and there is good evidence that man to man infection does not occur. However, it has been shown to be possible in animals, and we must be suspicious of hard and fast credos . . . I believe that infectious precautions should be used to prevent spitting and soil contramination, and I believe such hazards are special reasons for segregation and resection of cavity cases.

It is said that 75% of the *initial infections* are "subclinical"; 25% are "clinical", with a varying amount of illness ;and only ¼ to ½ per cent of the cases 'disseminate' thru the body in the white race . . . Darkskinned races tend to have endogenous progression, and negroes disseminate 10 times as often as whites . . . Dissemination is the great hazard, since there is a 50-60% mortality, and 100% mortality in fulminating cases. It is the result of a failure to focalize, and W. A. Winn says patients should be kept in bed until their lesions have focalized.

Symptoms may begin 1 to 3 weeks after exposeure. Primary pulmonary lesions are self-limited, may have 'flu'-like symptoms. Pulmonary infiltrates produce pain in about 80%, cough in 50%, fever in 60%, and arthralgia quite often. They usually last 2 weeks, though the patients may take months to convalesce. Sedimentation rates and the percentage of eosinophiles are elevated. A continued high sedimentation rate, a rising complement titer, and a loss of skin-test reaction suggest impending dissemination . . . In an endemic area, any malaise, bronchitis, fever, or x-ray shadow should be suspected.

Coccidioidin is a very specific skin-test antigen (available from Cutter Lab.). One may test with dilutions starting at 1:100 and continue down to 1:10. Only at 1:10 dilution does cross-reaction with histoplasmin occur . . . The antigen is made from a culture, like tuberculin, and the skin-test is similar to the Mantoux. It develops 2 to 4 weeks after exposure. It persists for years . . . Erythema nodosum or multiforme are also 'allergic' phenomena, and occur in about 20% of the cases at the same time as the skin-test hypersensitivity.

A *complement fixation* test also becomes positive as sensitivity develops. It indicates an active or progressive disease when positive and, as an indicator of humoral antibodies, it is transient. When it is negative or low, in the presence of a lung lesion it is either of no help or indicates focalization. It is so specific that it is said there are no false positives . . . The precipitation test indicates a recent infection, and supplements the complement fixation test to that extent. It does not persist long.

Cultural tests are hazardous to lab-workers. Sputum tests are not needed if diagnosis can be made by skin-test or serology . . . Specimens from lesions produce positive cultures in 55%, and from cavitation the incidence may be more than 90 per cent.

X-ray findings are variable, but the disease in humans usually localizes, usually in the lungs. Winn says that the first lesion is a patch of pneumonitis. Lesions in a group of soldiers included nodules, peri-bronchiel fibrosis, consolidation, hilar adenopathy (23%), cavitation (8%), pleural effusion (2.6%), and nodular dissemination. The initial cavity is usually thin-walled and closes spontaneously, though persistent cavities are notoriously hick-walled . . . In the series quoted, 100% had a history of exposure, 30% had erythema nodosum, 85% had an x-ray lesion, 80% had a positive skin-test, 86% a positive serology, 25% had excess eosinophiles, and 6% had a positive sputum.

'Cox and TB are concurrent enough so that Cotton could add his 24 cases to the 19 in the literature. He resected 9, of which 3 had positive serology and 5 had c. immitis in the sputum; they all did well. Eight infants and 7 adults (not resected) were all positive for TB by culture, and all but one were positive for 'cox' by serological tests and sputum culture.

The general rule is that in dissemination occurs after the acute phase, or focalization, and that there is no recurrence after resection, but rare cases of each *have* been reported.

The diagnosis of 'cox' is important not only because of the health of the patient, and the public health, and our pride, but because of the *medico-legal aspects* which Levan has described. It is becoming a terrific compensation problem for insurance companies, the courts, the industrial commission, and industries. Patients have been

compensated for seven different categories of exposure.

Discussion of Histoplasmosis

'Histo' is a currently fashionable topic. It was first described from autopsies in Panama 50 years ago; it was shown to be caused by a fungus, instead of protozoa in 1934; the cultural characteristics were reported in 1941; and there were less than 200 proved cases by 1952.

Cases have been reported in Central and South America, the Philippines, Java, South Africa, and England, with Panama well-known as a source. The chief North American endemic area encircles the junction of the Ohio and Mississippi rivers, with a decreasing incidence of cases in widening circles.

Definite *facts about infection and disease* are few. Several workers have found pulmonary calcifications in people with negative tuberculin reactions. The USPHS has made a "detective-like expose" since then. Furculow and Mantz tested 17,000 school children in Missouri with histoplasmin and found 72 symptom-free cases of 'histo'. A Texas survey of children showed 4.5% positive to tuberculin, 7% positive to histoplasmin. No certain source was known for such cases . . . Recent reports have shifted from endemics to *epidemics*. More than 20 epidemics have been described, and most of them carefully studied by skin-tests, serology, chest films, and soil cultures. They occurred in the states near Missouri, but as far north as Wisconsin, east to New York, south to Texas, and west to the Dakotas. They involved groups as small as families, and as large as 35 soldiers. . . . 'Histo' is known as a rural disease, with more men affected than women, though one suspects that this may be due to a greater amount of exposure of men to a 'reservoir' in the soil. The fungus apparently has a mycelial form in nature, and a yeastlike ovoid form in infected tissues, where it is usually intracellular and stains best with P.A.S. (periodic-acid-Schiff) stain. It selects the reticuloendothelial cells (or vice versa) and produces a granulomatous lesion which may caseate and then become encapsulated, calcify centrally, or arrely evacuate. The granuloma is somewhat like TB, 'Cox', brucellosis, sporotrichosis, kalazar, and sarcoid.

The *mode of infection* seems similar to that of TB and 'Cox'. The 'primary' involvement may produce a varying amount of illness. A pulmonary lesion produces the lung and chest symptoms which one would expect in about 50% of the cases. Dissemination is to be feared, since it is highly fatal, but it is very rare. Old people are supposed to suffer most, and to have mucocutaneous lesions, but since the 'classic' picture of routine generalized involvment is now said not to be so, we may have to change our conception as we learn more.

Claggett reported 20 cases of the *disease* from the Mayo Clinic. Nineteen were diagnosed by culture from a dozen tissue sites. Most had fever and fatigue; 14 had chest lesions; 7 had oropharynegeal, liver and spleen lesions; 6 had adrenal involvment; and 7 had peripheral node enlargement. There were 11 deaths since diagnosis. Renal and adrenal lesions had the worst prognosis.

Epidemics have regularly been found to be associated with histoplasma in specimens of soil. Random dirt samples on farms were 1.6% positive but chickenyard dirt was 13.7% positive, and soil from bat caves, cowbarns, basements, and birdroosts was also highly contaminated . . . Dogs and rodents may develop the disease, but fowl rarely have infection or antibodies. Their role is uncertain.

The *lung lesion in endemic cases* seem to have their own x-ray characteristics. Two-thirds of the 72 cases found in the large St. Louis survey had sharply circumscribed nodules, and 25% had patchy irregular lesions. Occurrence of calcification is known to be central and to occur slowly. The older lesions often have a somewhat 'target' appearance, with the density decreasing from the 'bullseye' to he periphery . . . Another series of 65 cases had only 4 apical lesions (like reinfection TB), and only 3 cavities.

Skin-tests with histoplasmin are of uncertain value in individual cases. The test becomes positive 2 to 4 weeks after infection. There were only sixteen positives in a series of 31 known chronic cases, but 94 of 95 acute epidemic cases had positive skin-tests. The answer must lie in the fact that anergy developes gradually during the 5 years after infection, and in many 'disseminated' cases. The antigen is newly available from Parke Davis and Co. A standardized volume is diluted by 1 cc. of diluent, and a single dose of 0.1 cc. is given intradermally. An induration of 5 mm. or more is a positive reaction.

Bacterial studies of sputa are rarely positive, and in one series only 1 of 20 cases had a positive culture. Diagnosis can be made by biopsy,

but tissue cultures may help differentiate from toxoplasmosis, cryptococcus infection, Leishmaniasic, and one form of blastomyces.

Complement fixation is also an equivocal test. It remains positive for about a year. Dilutions of 1:40 or more are significant, yet in 17 cases of lung disease proved by resection 9 were negative and 8 were positive in the range of from 1:5 to 1:30 . . . There is also some cross-sensitivity with other fungus infections.

Data on 34 resected cases showed that 28 were male and 40% had lived in Panama. Thirty cases were skin-tested for 'histo', 'cox', and TB; 7 were positive to all three, 3 were positive for the fungi, 13 were positive for 'histo' and TB, 6 were positive only for 'histo', and one was positive only to tuberculin.

Thirty five *'round focus' lesions* examined at the Army Institute of Pathology had no known cause in 8, TB in 6, 'cox' in 3, and histoplasmosis in 18. 'Histo' is a more than ordinarily considered cause of such lesions . . . Other series of solitary dense lesions are found to have a neoplastic cause in 25 to 30 percent, and two recent reports on the value of the periodic acid-Schiff stain substantiate the findings of the Army study that 'histo' and 'cox' can be found by that method in a surprising percentage of cases.

It would seem logical to be highly suspicious of any pulmonary symptoms and of all x-ray lesions until the etiology has been decided. One should push ahead to a diagnosis if at all possible . . . Chemotherapy is apparently not yet effective, but better drugs can be expected and, when it seems legitimate, used in a controlled way . . . Excisional surgery should be considered for destroyed or cavitative pulmonary lesions, and infectious disease precautions used until the potential source has been eliminated . . . There is bound to be better understanding of fungus diseases, even in the next year or so, and we can both profit and help.

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HIRSUTISM

A resume of etiology and current therapy.

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HIRSUTISM is not welcomed by any girl or woman at any age, particularly abnormal facial hair. She becomes less feminine and more masculine in many ways, none of which increases her charm nor makes her happy. The daily chore of making herself attractive becomes an unwelcome task a part of which is the nuisance of shaving face, leg, and body hair. Such abnormal hair growth is with few exceptions evidence of a hormone imbalance. No matter what the basic cause of the endocrine dysfunction eventually proves to be, an absolute or relative excess amount of 11-corticosteroids or other hormones secreted by the adrenal cortex or of testosterone or of both, in conjunction with a relatively low serum estrogen level is the apparent factor which induces hirsutism in women (1) (2) (4).

An accurate diagnosis of the cause of this hormone imbalance in an individual is imperative before medical or surgical therapy can be intelligently prescribed. Study of thyroid and ovarian function must be correlated with adrenal and anterior pituitary function tests before any therapy is started. The longer the delay in starting therapy or the longer the hirsutism has existed, the more difficult is its cure or control. The important point is that most women at any age can now be cured of hirsutism or the condition kept under reasonable control with hormone therapy now available. Any woman who has been obliged to shave daily is very grateful when her abnormal hair growth has been controlled.

A list of the causes of hirsutism is here presented as an aid in the discussion of the problem:

A. Ovarian
   1. Sub-normal ovarian function.
   2. Non-functioning ovaries – the true menopausal cessation of function.
   3. "Cystic" ovaries and polycystic ovaries.
   4. Malignant and other tumors of the ovary.
   5. Congenital absence of ovaries.

B. Adrenal Cortex
   6. Congenital benign adrenal cortex hyperplasia.
   7. Benign hyperplasia of the adrenal cortex.
   8. Benign adenoma of the adrenal cortex.
   9. Carcinoma of the adrenal cortex.

C. Anterior pituitary
   10. Tumor, benign or malignant of the anterior pituitary gland.

D. Medication
   11. Testosterone.
   12. Cortisone and ACTH.
   13. Estrogen.

E. Surgical
   14. Hysterectomy.
   15. Removal of one ovary.
   16. Castration.

F. Idiopathic

G. Rare

1. SUBNORMAL OVARIAN FUNCTION

Fortunately for women in the period from adolescence until the climacteric poor ovarian function is the most frequent cause of the mild hirsutism frequently complained of to the family physician. Few physicians, parents or even husbands realize how many young women shave excessive hair growth on their legs. Many girls resent upper lip hair but do nothing about it. A considerable number shave regularly, a fact unknown by intimate friends and even their immediate family.

A few have hair removed by electrolysis.

Therapy regularly helpful to stimulate ovarian function and thus relieve hirsutism is thyroid extract grain ½ together with stilboestrol 0.1 mg. daily. If some improvement is not noted in three or four months, stimulation of anterior pituitary, thyroid (16) and ovarian function with pregnant mare serum hormone, eg., Gonadogen (Upjohn) regularly corrects mild hirsutism and other complaints. Response is slow but improvement continues for months. In younger girls cure is often attained.

It is often difficult to prove whether there is deficient FSH or LH stimulation (or both) of the ovary. Inability of the ovary before the natural menopause to respond to adequate anterior pituitary trophic hormone stimulation is, in my opinion, rare. I prefer to state that abnormal thyroid or adrenal cortex or both hormone secretions can block ovarian response to FSH and LH stim-

ulation by disturbing balanced anterior pituitary function.

2. NON-FUNCTIONING OVARIES.

Women in natural menopause who have gone into uneventful change of life uncomplicated by other endocrine pathology, are very commonly annoyed by excess chin and lip hair. There is now available a potent, long acting estrogen, Depo-Estradiol (Upjohn), which is very effective in controlling hirsutism as well as many unwelcome menopausal complaints sometimes resistant to therapy with other estrogen, oral or injectable. It appears that in menopausal women absolute estrogen deficit in itself predisposes to excess facial hair growth perhaps because of the relative hormone imbalance between the adrenal and ovarian hormones. Even though the adrenal hormone blood level is normal, the serum estrogen level in the true climacteric woman is regularly comparatively low. Since by definition the menopausal ovary is nonfunctioning due to natural aging, substitution therapy using estrogen in amounts adequate to relieve the complaints is indicated. Stimulating therapy is not indicated because the ovary cannot respond to anterior pituitary trophic hormones.

3. "CYSTIC" OVARIES AND POLYCYSTIC OVARIES.

What constitutes a diagnosis of cystic and polycystic ovaries? (1) (11) (12) (13). Too many ovaries are removed or mutilated because one or several cysts smaller than a lemon are found on an ovary during laparotomy. Such cysts can be punctured and the ovary left alone. Isolated cysts larger than a lemon may be excised, but the ovary should not be removed. (17)

Hirsutism, amenorrhoea and sterility are regularly associated with huge polycystic ovaries but rarely with the ordinary "cystic" ovary. Wedge resection of large polycystic ovaries (usually the procedure is performed on both ovaries) is a compromise with total removal and is justifiable. The operation may cause remission of symptoms for a few months in that menstruation may be resumed, but seldom is there much permanent improvement. Such "mentsruation" is usually estrogen withdrawal bleeding and does not become regularly cyclic. After several episodes of vaginal bleeding amenorrhoea commonly recurs. The hirsutism and sterility generally persist. While surgical cure is rare, stimulating endocrine therapy following wedge resection is today worth trying.

Failure of ovulation (rupture of the mature ovarian cyst) which is possibly the cause of ordinary "cystic" ovaries, might be due to lack of adequate anterior pituitary follicle stimulation hormone (FSH). Why the immature ovarian follicles fail to regress is unknown, but inadequate luteinizing hormone (LH) stimulation could be a factor. It is now well known that there are four important anterior pituitary trophic hormones which must be secreted in adequate balanced amounts to insure radiant feminine health:—

1. FSH which stimulates ovarian follicle growth and hence estrogen secretion by the ovary.
2. Luteinizing hormone (LH) which stimulates corpus luteum formation following ovulation following ovulation and hence secretion of progestin.
3. Thyroid stimulating hormore (TSH) which stimulates the thyroid gland to convert serum iodine into thyroxine as measured by the serum protein-bound iodine procedure.
4. Adrenal-cortico-trophic-hormone (ACTH) which stimulates the adrenal cortex to secrete cortisone and the cortisone-like adrenal steroid hormones.

When a woman is suspected or known to be not ovulating, there is usually clinical evidence of inadequate FSH secretion as well as inadequate imbalanced secretion of one or more other anterior pituitary trophic hormones. Therapy should therefore logically be directed at creating balanced hormone secretion by stimulating anterior pituitary function thus returning the thyroid, adrenal cortex and ovaries to normal function and normal balanced secretion of their hormones. With the hormones and diagnostic procedures now available, much can be done to relieve the hirsutism, prevent "cystic" ovaries by correcting their cause and abolish other associated symptoms. Basic therapy is the same as that for stimulating subnormal ovarian function. Therapy for polycystic ovaries following wedge resection is the same but in addition the suppression of excess ACTH secretion is usually a problem.

4. MALIGNANT AND OTHER TUMORS OF THE OVARY.

Rarely are tumors of the ovary encountered which masculinize a previously normal feminine woman by causing marked hirsutism, among other changes. This is an entirely different diagnostic problem than those just discussed. All

such masculinizing tumors of the ovary are not hopeless carcinomas. Therapy of malignant and other tumors of the ovary is prompt surgical removal about which there should be no controversy(1).

5. CONGENITAL ABSENCE OF OVARIES.

Congenital absence of both ovaries is rare but has been reported as a cause of hirsutism (14).

6. CONGENITAL BENIGN ADRENAL CORTEX HYPERPLASIA.

Congenital benign hyperplasia of either or both adrenal cortical tissue in young girls is the most common cause of female pseudohermaphroditism (8) (9). The individual seems to have been born a girl but her growth and sexual development make her masculine. Much can be done today for these unfortunate girls whose medical management is often difficult (10). Hirsutism is only one of their problems. Therapy is preferably started before adolescence. The diagnostic difficulties are often great and since considerable expense is inevitable, the economic problem often prevents or delays diagnostic study and therapeutic measures. Delay may cause the condition to advance until confusion exists as to the actual sex of the individual. Then referral to the large teaching institutions as charity patients is sensible and often the only chance to make life bearable for her.

7. BENIGN HYPERPLASIA OF THE ADRENAL CORTEX.

Benign hyperplasia of the adrenal cortex in the mature woman is an uncommon cause of hirsutism and other complaints. (3) (4). Rarely is hirsutism alone the symptom for which a woman seeks relief. While severe degrees of hyperplasia can usually be accurately determined, the mild degrees are difficult to detect. Laboratory measurements of serum steroid hormone levels are not generally available despite a tremendous amount of research (5). To quote Gardner, ". . . there is considerable evidence that the normal ovary produces androgen . . . as yet no adequate data seems to be available to indicate whether it is adrenal cortex or ovary that is hyperfunctioning in patients with idiopathic hirsutism. Improved techniques for the estimation of pituitary trophic hormones such as LH may provide the answer to the question."

The same problems of hormone assays for diagnosis are met in benign hyperplasia of the adrenal cortex as in idiopathic hirsutism. Blood serum assays for estrogens, androgens and adrenal cortex hormones which could be performed at reasonable cost by a local laboratory would be a tremendous help.

It is now generally known that cortisone and chemically similar adrenal hormones given in adequate dosage to control the collagen disease for which they are commonly used, will suppress ACTH secretion by the anterior pituitary. Such a condition is desirable as therapy for many medical purposes including benign hyperplasia of the adrenal cortex. Removing constant ACTH stimulation of the adrenal cortex sooner or later leads to partial or complete atrophy of the adrenal cortex which in itself is a potentially dangerous and at times a difficult condition to control. In an occasional severe form of hirsutism due to benign hyperplasia of the adrenal cortex such drastic medical therapy might be justified.

Total or sub-total surgical removal of both adrenals can be successfully performed today but will probably seldom be done for benign adrenal hyperplasia solely to cure hirsutism (6).

8. BENIGN ADENOMA OF THE ADRENAL CORTEX.

Hirsutism may rarely be due to a benign adrenal adenoma which causes the same clinical picture as Cushing's syndrome. Therapy is prompt surgical removal of the adrenal tumor as soon as the diagnosis can be accurately made. However, if tumor or carcinoma of the pituitary is causing adrenal cortex hyperplasia, no benign adrenal tumor will be found at operation. Accuracy in diagnosis is imperative to prevent useless surgery (1) (4) (7) (15).

9. CARCINOMA OF THE ADRENAL CORTEX.

When hirsutism and Cushing's syndrome are due to carcinoma of an adrenal gland, early operation is imperative. Such a growth can be present for months and not spread beyond the capsule of the adrenal gland. Fortunately, only one gland is usually involved. If surgery is not delayed too long, the hirsutism and the cancer is often cured (7). Diagnosis is not as difficult as formerly (1) (4).

An excellent comment on the differential diagnosis of adrenal cortex pathology is ably summarized by Laidlaw et al., (4) which is quoted in its entirety:—

"The clinical picture of hyperadrenocorticism

extends in a broad spectrum from Cushing's syndrome at one end to adrenogenital syndrome at the other. When the patient presents one or more elements of Cushing's syndrome, attention is focused on the adrenal cortex wherein may be found bilateral hyperplasia, adenoma or carcinoma. These lesions may be differentiated by the response of the urinary 17-hydroxycorticiods to ACTH. Patients with bilateral hyperplasia show a hyperactive response to ACTH; those with carcinoma do not respond. In three patients with adrenocortical adenoma, a normal response to ACTH was obtained; a fourth showed a hyperactive response The degree of automy of the adenoma probably determines the response of the tumor to ACTH stimulation.

"In contrast to Cushing's syndrome, the virilization syndrome may be caused by a lesion in either the adrenal cortex or the ovary. In such cases, a course of cortisone administration is instituted. If a dramatic fall in urinary 17-ketosteroids occurs, the causative lesion is presumed to be bilateral adrenal hyperplasia. In the absence of depression of 17-ketosteroid excretion, the lesion is considered to be either an adrenocortical tumor or a lesion in the ovary. In these instances, pelvic laparotomy is first performed. In the absence of a responsible ovarian lesion, the adrenals are then explored."

10. TUMOR OF THE ANTERIOR PITUITARY.

Cushing's disease is a comparatively rare disease complex in which an anterior pituitary tumor causes an otherwise normal adrenal cortex to become hyperplastic. Cushing's disease presents the same clinical picture as Cushing's syndrome only the cause is different. Fortunately X-therapy to the pituitary growth is now regularly successful and surgical removal of the pituitary gland is less frequently indicated (15). No adrenal surgery is indicated since adrenal cortex function is normal, but the glands are hyperplastic due to hypersecretion of ACTH by the anterior pituitary tumor. If irradiation is successful the adrenals resume normal function and the hirsutism and other symptoms of Cushing's disease gradually regress.

11. ANDROGEN MEDICATION.

When androgen medication is given to women, often in combination with estrogen for menopausal symptoms or osteoporosis, excess facial hair growth often soon becomes a problem despite relief from the complaint for which it was administered. In such cases stopping the androgen, commonly testosterone, is imperative and sufficient to stop the hirsutism (1).

12. CORTISONE AND ACTH MEDICATION.

The facial hair growth and typical edema of the face which commonly follows therapy with cortisone tablets is well known and today is usually recognized promptly (7). This type of hirsutism usually regresses slowly when the ACTH or cortisone therapy is stopped. If the medication is resumed all the symptoms of Cushing's syndrome will recur unless the dose is carefully adjusted to individual needs. For the occasional patient who has great difficulty taking urgently needed cortisone or ACTH for some disease without causing hirsutism and other undesirable side effects, other adrenal hormones are now available (3) which have a cortisone effect but are less likely to cause some of the complications. At times by combining an estrogen with a bile acid and salicylates, a potentiating effect is secured so that a smaller dose of cortisone is effective therapeutically.

13. ESTROGEN MEDICATION.

Large dosage of estrogen when functioning ovarian tissue is present will depress ovarian function by suppression of anterior pituitary FSH secretion. It is entirely analogous to cortisone effect on adrenal cortex function and thyroid extract on thyroid gland function. Ovarian function depressed or abolished by estrogen medication may cause hirsutism or may aggravate any hirsutism already existing. It should be evident that estrogens are NOT helpful in therapy of hirsutism in mentruating women. Stimulating therapy IS helpful.

14. HYSTERECTOMY.

A young woman regularly notes the onset of facial hair following hysterectomy even when both ovaries have been retained. It is a clinical fact that hysterectomy alone depresses ovarian function in many women frequently causing menopausal-like symptoms for which estrogens are often mistakenly given. Therapy to stimulate ovarian function is indicated.

15. REMOVAL OF ONE OVARY.

The common surgical practice to remove one ovary, while removing an appendix or performing other surgical procedure in a young woman because of "cysts" is not an innocuous procedure. It accomplishes little or nothing constructive. Following the operation all too frequently hirsutism and menopausal-like complaints become serious.

Even when hysterectomy has not been performed, the remaining ovary is often unable to carry the burden without help from stimulating therapy. When both ovaries are present, response to therapy is more abundant health.

16. CASTRATION.

Estrogen substitution therapy is necessary if castration has been performed prior to the natural menopause whether by surgery or irradiation. Therapy is usually much more complicated and unsatisfactory in the young woman than the simple estrogen substitution therapy of the natural climacteric. Therefore, when, during laparotomy in the premenopausal woman, ordinary "cystic" ovaries are found, it is suggested that the ovaries not be removed nor wedge resection performed. Puncture the large cysts leaving the chocolate cysts and any patches of endometriosis not readily accessible. With medical endocrine therapy now available or under investigation, many of these women can be returned to normal ovarian function. Such is possible only when the ovaries have been retained. Castration is an irrevocable tragedy. Few of these women are psychoneurotic. Their complaints are truly problems of ill health due to hormone imbalance.

F. IDIOPATHIC HIRSUTISM.

The pronounced black facial and body hair growth commonly afflicting women of the Mediterranean countries is possibly an idiopathic (1) or hereditary condition rather than an endocrine dysfunction. These women do not usually have a condition defined as hirsutism which is regularly encountered (5) with endocrine abnormalities. Some of these women can be helped by appropriate therapy.

G. OTHER CAUSES OF HIRSUTISM are medical curiosities rarely encountered.

Hirsutism is a complaint common to all these disorders. Some require prompt surgery. Others prolonged hormone therapy. An accurate diagnosis is imperative and the various possibilities should be constantly kept in mind. Never dismiss a woman with the statement that nothing can be done to relieve her abnormal facial hair growth. Today much can be offered in the way of control or cure if only she will co-operate in the diagnostic procedures and can afford the hormone assays and therapy.

In summary then, when hirsutism becomes annoying at any age the chance of a woman having a carcinoma or tumor of an endocrine gland is relatively small. Hormone imbalances readily amenable to endocrine therapy cause most hirsutism. Today there is medication available which will cure or readily control the mild degrees of obnoxious hair growth which commonly annoy women.

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SPINAL ANESTHESIA CAN BE SAFE*

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SPINAL anesthesia can be safe if it is administered by a physician who is well versed in the fundamental principles of anesthesiology and has a basic knowledge of the anatomy and physiology of the central nervous system. He must be familiar with the pharmacology and dosage of the agents used and should be cognizant of the indications, contraindictions and complications of the method employed. This physician must finally be willing to minutely observe the patient from the time of induction to the end of the procedure. These prerequisites for a safe spinal anesthesia are mandatory.

A physician's basic knowledge of the fundamental principles of anesthesiology is not enough in itself to guarantee the safe conduct of a spinal anesthesia. He must honestly and sincerely put these principles into practice. For example, it is not enough to know that a patent airway with adequate ventilation is necessary at all times. The physician must be astute enough to recognize minute degree of obstruction and depressed respiratory exchange and then be technically competent to manupulate the mandible in such a way that a free and open passageway exists from the external nares through the pharynx and glottic opening into the trachea. Blithely going through the motions of holding up the chin without accomplishing this objective is idiotic, tiring to the physician, and ultimately harmful to the patient. It is even worse not to recognize and correct the performance of this useless procedure. If the simple expedient of holding the chin does not create an open airway, then the physician must be able to properly insert a mechanical airway, be it metal, rubber or plastic. If the above does not suffice, then he may have to resort to the placement of nasopharyngeal or endotracheal tubes. The physician must both master the technical dexterity to place all these artificial aids, and possess the intelligence to recognize whether these aids are performing the functions expected of them. The mere fact that an airway is in the mouth or an endotracheal tube has been inserted does not in itself guarantee an open passageway. For instance, the base of the tongue may still be flush against the posterior pharyngeal wall due to slipping forward of the oral airway. Likewise, the endotracheal tube itself may become kinked or even inserted deep into a bronchus. These conditions must be recognized immediately and corrected.

If one wonders why this fundamental demand for a patent airway is stressed as a prerequisite for safe spinal anesthesia, it is because most deaths under spinal anesthesia are due to asphyxia. These deaths occur when the effects of premedication are so profound as to put the patient into surgical anesthesia, or when an intraventous barbiturate has been given to facilitate the introduction of the spinal needle or alleviate traction reflexes during the course of the operation. Any noisy breathing means obstruction and must be eliminated at once. Patients under the influence of depressant drugs are not merely snoring. They are obstructed.

Asphyxia also occurs when the spinal anesthesia level creeps cephalad and paralyzes the intercostal nerves. The phrenic nerves and medullary centers may also become involved. A fundamental principle of anesthesiology states that postoperative complications vary directly as the length of time of the operation and the depth of the anesthesia (or in spinal anesthesia, the height). Asphyxia associated with a high spinal well illustrates this law. Should the physician, even though aware of these basic truisms, be so unfortunate as to have asphyxia occur in his patient, he must be prepared to administer artificial respiration. This means that besides being able to establish a patent airway, he must be capable of ventilating the patient with oxygen and maintaining a normal circulation at the same time. Cerebral damage or cardiac arrest is the price to be paid should the physician fail.

A knowledge of the anatomy and physiology of the central nervous system, particularly the spinal canal and cord, is essential for the giving of safe spinal anesthesia. The physician should be familiar with the innervation of the part to be anesthetized. The nerve supply of any structure actually consists of three components — sensory,

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motor and sympathetic. It is not always enough to get a somatic sensory level of anesthesia adequate for the skin incision if the visceral afferent sympathetics from the area are also to be stimulated. This duel innervation is particularly seen in lower extremity surgery where a tourniquet is employed and in hernia operations where there is marked traction on the spermatic cord. Stimulation, by a tourniquet, of the sympathetics accompanying the blood vessels in a lower extremity may cause extreme discomfort to a patient even though the spinal anesthesia has blocked out all incisional pain. Profound drop in blood pressure has occurred during a herniorrhaphy when traction has been applied to the cord even though the rest of the operation has been uneventful under a supposedly adequate spinal anesthesia. This is because the sympathetic nerves enter the cord at a higher level than the corresponding dermatomes. Thus a higher level of spinal anesthesia is required when the sypmathetics are involved. Likewise, the degree of medullation of spinal nerve fibers is important in determining the amount of drug taken up by these fibers. This in turn makes for a more or less profound spinal anesthesia. It is necessary to remember that the dorsal or sensory roots lie in the posterior portion of the spinal canal while the motor or ventral roots emerge from the anterior portion of the canal. The preganglionic fibers of the sympathetic pathways arise in the intermediolateral horn of the gray matter of the thoracic and upper lumbar cord. Since spinal anesthetic drugs exert their effect not only on the roots but probably penetrate the cord as well, we can profitably apply this knowledge of anatomy to help accomplish a safe and satisfactory spinal anesthesia. The greatest concentration of the drug should be deposited on the dorsal roots and posterior portion of the cord with very little drug bathing the motor components. This deposition of drugs results in satisfactory anesthesia and protects the patient from the danger of intercostal or vasomotor paralysis should the anesthetic level reach the thoracic segments. The curves of the spinal column and the presence of the dentate ligaments are important anatomical barriers to keep in mind as they effect the height to which the drugs may ascend. A knowledge of the angle of inclination of the spinous processes at various levels determines the ease with which a spinal tap is accomplished.

The physiology of the central nervous system is as important as its anatomy. Unless the physician understands the functions of the autonomic nervous system he will be at a loss to explain the hypotension, bradycardia, constricted bowel, or epinephrine depletion of the adrenal gland in a patient under spinal anesthesia. For the same reason it is necessary to know the physiology of the cerebrospinal fluid, its site of formation, absorption, composition, amount, pressure, circulation, pH and specific gravity. For example, if one is not aware that the specific gravity of spinal fluid varies between 1.003 and 1.009, then one cannot make the proper selection of an anesthetic drug with the optimum baricity (hyper, hypo, or isobaric) in relation to the specific gravity of spinal fluid. The fact is well established that the more a patient is moved during the early phases of a spinal anesthesia the more chance there is of creating a hypotension. This can be eliminated by selecting an agent with the proper baricity which will immediately gravitate to the dorsal roots as the patient is being positioned for surgery. In this way the agent is "fixed" in the final operating position and no further turning of the patient is necessary. Thus a minimal involvement of the motor and sympathetic components occurs.

A physician should make no attempt to administer a spinal anesthetic unless he is completely acquainted with the pharmacology and dosage of spinal anesthetic agents. It can only lead to disaster. Specifically, the physician should be aware that spinal anesthetic drugs have various chemical configurations and are for the most part nitrogenous derivatives, falling into an ester and a miscellaneous group. Knowing this he is better equipped to select an alternative drug should the patient give a history of a specific drug idiosyncrasy. The ester group is by far the largest and includes most of the drugs in current use today. They are procaine, tetracaine, and piperocaine. Drugs in the miscellaneous group are not esters but compound products. Dibucaine is an example of a drug of this classification and being a quinoline derivative, it has a different chemical configuration than procaine and so can be used if there is a history of procaine sensitivity.

Selection and administration of the proper agent for the operation to be performed presupposes knowledge of several factors, one of which is the position in which the patient will be placed. Only in this way can a drug with the right

baricity be administered. A preoperative knowledge of the length of time it will take to do the proposed operation will help in determining the agents to be used, whether adrenalin should be added, or the continuous method employed. Another factor to be considered is the dosage to be employed. This should be calculated by exact weight of the drug given, not by ampules. The dose should be considered as so many milligrams of tetracaine and not as one ampule or 2 cc. The solutions injected into the spinal canal should also be at an optimum pH otherwise precipitation occurs. If the solutions are not at the proper concentration, nerve damage ensues. Knowledge of the rapidity of action of the anesthetic drugs as well as the time of their fixation will determine when surgery can start and when it is safe to radically change the patient's position. Placing a patient in a steep Trendelenburg position before a hyperbaric spinal anesthetic has had time to set may be fatal. In general, it can be said that the level to which a spinal anesthesia ascends depends on the following five factors:

1. Baricity and position of the patient.
2. Length of the spinal canal and anatomical variations in the normal spinal curvature.
3. Volume of the anesthetic solution used — the larger the volume the higher the level of anesthesia obtained.
4. Concentration of the agent.
5. Rate of injection of anesthetic drug — the faster the injection, the higher the level.

Knowledge of the average duration of action of anesthetic agents will help the physician to plan supplementation should the operation run over the estimated time. This knowledge should be augmented with the further knowledge that the sensory fibers are the first to be anesthetized and the last to recover. The sympathetic fibers are the next to be involved and may recover either second or first. The motor fibers are the last to be affected and are usually the first to recover. This information is of practical importance in the administration of a continuous spinal where supplemental doses must be given before the spinal anesthesia wears off. When this does occur, it is often difficult to reestablish a satisfactory level of anesthesia and larger dosages of supplemental drugs are required. Because of the effect of the agents on the motor and sympatheic fibers, one can be on the alert and recognize the return of motor tone, perspiration, rise in blood pressure, and restlessness as precursors to the return of sensory activity. Appropriate action can then be taken before a crisis occurs. It is also well to know that spinal anesthesia wears off from above downward. The perineum is the last dermal area to recover.

With this basic information at hand the physician should now consider the indications and contraindications for a spinal anesthesia and then weigh the advantages obtained from its administration against the complications that can occur. In general it can be said that unless there is a definite contraindication (these will be discussed later) against spinal anesthesia, this method can safely be used for anyone. Where there is a choice of several methods of anesthesia, certain relative indications for a spinal technique make this the method of choice. The indications can be listed as follows:

1. All operations below the umbilicus unless, as mentioned above, there is a definite contraindication to spinal anesthesia.—This does not mean that spinal anesthesia cannot be used for operations in the upper abdomen, chest, or even the neck. It has been used, and quite satisfactorally, for thoracoplasties and even thyroid surgery. It does mean that when the level of anesthesia ascends above the umbilicus the complications are increased and the safety factor is decreased. So, unless one can justify the added risk, it is much wiser to confine spinal anesthesia to lower abdominal and extremity operations.

2. Patients in whom difficulty in maintaining an airway is anticipated.—In these cases it is expedient to have the patient responsible for his own respiratory ventilation. This situation can occur with patients in bizarre operaing positions or when, because of an anatomical anomaly or pathological process, it is impossible to pass an endotracheal tube. If the operative site is below the umbilicus in these patients then spinal anesthesia may be more efficacious than an inhalation or intravenous anesthesia.

3. Relaxation. — Even though spinal anesthesia may give maximal relaxation this is a relative indication, as satisfactory relaxation can also be obtained with curariform drugs. Spinal anesthesia in addition to relaxing the abdominal musculature also constricts the intestines, thereby allowing more operating room in the abdominal cavity.

4. Presence of liver or kidney disease.—Spinal anesthesia causes no significent change in liver

function and does not affect glomerular filtration, tubular reabsorption, or urinary secretion unless hypotension or hypoxia are permitted to supervene.

5. Presence of certain metalolic diseases, such as diabetes.—There are no significant blood chemical changes under a well-managed spinal anesthesia. The carbon dioxide content of the blood is unchanged if respiration is not depressed. Likewise, the carbon dioxide combining power is not affected and the blood glucose is unaltered.

6. Presence of explosion hazards.—Spinal anesthesia allows the use of cautery and electrical appliances during surgery.

As can logically be seen, the advantages of spinal anesthesia depend on the absence of any contraindications. Contraindications are either absolute or relative. The only absolute contraindication is infection at the site of the lumbar puncture. It is obvious that no physician would deliberately introduce infection into the spinal canal.

The relative contraindications are:

1. Debilitation and shock.—The presence of a low blood volume in these cases makes spinal anesthesia dangerous. If hypovolemia is compensated and the involvement of the sympathetic component corrected, then spinal anesthesia can be given relatively safely. A continuous or fractional administration of spinal agents in these cases allows for better control.

2. Presence of severe hypertension or hypotension.—Here again it is not the spinal drugs themselves that are dangerous but the inability of the physician to manage the circulatory affects the agents produce by their action on the sympathetics.

3. Extremes of age and very apprehensive patients.—Although spinal anesthesia has been administered to all age groups, one must be aware that the very young and very old, like the apprehensive patient, may be psychically unsuited for this method. It is folly to administer a spinal anesthesia unless rapport exists between the patient and the physician.

4. Presence of respiratory disease.—Spinal anesthesia does not decrease the incidence of postoperative pulmonary complications and in some instances may increase their number. Partial intercostal paralysis resulting from a high spinal may well set the stage for an atalectasis. In patients with marked pulmonary secretions, an inhalation endotracheal technique can be employed for the primary purpose of performing a tracheal toilet.

5. Presence of intraspinal lesions or infections—If the pathological process in a spinal cord is arrested, as in post-poliomyelitis or traumatic transections, the administration of spinal anesthesia will do no further damage. If the spinal tap is purulent or persistently bloody, anesthetic drugs should not be injected.

6. Intestinal obstruction. — Since there is increased activity, tone and constriction of the bowel following administration of spinal anesthesia, perforation of a distended loop of gut adjacent to a mechanical obstruction can result when this method is used. Conversely, an adynamic ileus may be cured by spinal anesthesia.

In contemplating the administration of spinal anesthesia, the physician should be alert to the complications that can occur. These fall into two categories — the immediate and the delayed. The immediate reactions consist of:

1. Bloody spinal tap. — A bloody tap may be an indication of intrinsic central nervous system pathology or evidence of traumatic insertion of the spinal needle. If due to the latter cause, the spinal fluid will usually clear readily and it will be safe to administer the anesthetic drug. Should the bleeding persist, it is wiser to abandon the procedure and use some other method of anesthesia. The presence of an excess of blood in the canal may result in an unsatisfactory anesthesia because of the altered pH and specific gravity of the spinal fluid.

2. Too high an anesthetic level. — If intercostal paralysis occurs, resuscitative measures must be employed until the effects of the agent are dissipated. As mentioned above, these measures consist primarily of administration of oxygen under pressure, and fluids to maintain normal circulating volume. Atropine may be indicated to counteract some of the vagal effects that occur when the cardio- accelerator fibers are involved due to a high level of spinal anesthesia.

3. Hypotension. — This is usually associated with a high anesthetic level and is accompanied by peripheral vasodilation and bradycardia. It thus differs from shock which presents peripheral vasoconstriction with tachycardia. The use of a vasoconstrictor agent prior to administration of a spinal anesthesia will often prevent the immediate spinal hypotension, which of itself is not harmful if the hypotension is not too low nor excessively prolonged. Hypotension can easily

be converted into shock by unrecognized blood loss or sustained traction reflexes. Hypotension during spinal anesthesia should be recognized for what it is and appropriate treatment instituted.

4. Nausea and vomiting. — This complication can occur at any time during the course of spinal anesthesia and from a variety of causes. Traction reflexes, hypotension with hypoxia, apprehension, and drug reactions have all been implicated. Proper treatment depends on a correct interpretation of the etiology.

Delayed complications are:

1. Cephalgia. — not every headache occurring after spinal anesthesia is caused by the anesthesia. Postspinal headache is a typical and distinct entity. It is probably due to decreased intraspinal pressure from leakage of spinal fluid through the opening made by the spinal needle in the dura. This complication is the most prevalent reaction occurring after spinal anesthesia and can at times become incapacitating. Treatment is aimed at correcting intraspinal pressure. The incidence of spinal headache can be reduced by paying attention to the following details: having the patient well hydrated before operation, meticulous attention to the lumbar puncture, prevention of extreme hypotension, and the use of oxygen during the operation. Even without treatment all postspinal headaches will eventually disappear. The cause of other postoperative headaches should be searched for and eliminated.

2. Meningismus. — Some patients will complain of stiffness or pain upon flexing the neck after a spinal anesthesia. Mild changes in the spinal fluid may be found but a culture is negative. Headache is usually present. These symptoms are self-limited.

3. Meningitis. — This can either be a pachymeningitis or leptomeningitis. A frank, purulent, specific meningitis can occur and must be differentiated from a meningismus if appropriate treatment is to be instituted.

4. Nerve injuries. — Abducens palsy has been seen following spinal anesthesia as well as after administration of other types of anesthesia. Cauda equina syndromes have occurred and paresthesias in other specific nerves have been reported.

5. Paraplegia and quadraplegia. — These most dreaded complications are probably on a chemical basis and are caused by physiological transection of the cord by contaminents in the anesthetic solutions used. Prevention of contamination is the only answer to this problem.

6. Backache. — The local tissue trauma and hematomas resulting from the unskilled insertion of a spinal needle are, in many instances, responsible for this complication. The stretching of ligaments and muscles when placing patients in adverse positions under spinal anesthesia may also account for many of these complaints.

7. Hysteria and malingering.

Everything that has been discussed so far to make spinal anesthesia safe can be found in standard textbooks.(1,2,3) The final safeguard to secure the safe conduct of spinal anesthesia lies within the physician himself. It is the willingness to stay with the patient throughout the operation and minutely observe and correct any changes in the patient's physiology before these conditions progress and result in catastrophe. To do otherwise is gross negligence with all its ugly implications.

SUMMARY

An attempt has been made to present the factual knowledge necessary for any physician to have if he wishes to perform safe spinal anesthesia. A plea is entered to have this physician stay with the patient throughout the procedure rendering minute to minute care until the operation is completed. In conclusion it is well to remember that a spinal anesthesia is only as safe as the knowledge and ability of the physician administering it.

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PHOENIX Clinical CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

MASSACHUSETTS GENERAL HOSPITAL

PRESENTATION OF CASE 37442

A forty-seven-year-old man was admitted to the hospital in an unresponsive and incoherent condition.

One week prior to admission he began to complain of general malaise. On the following day he developed a severe cough productive of large amounts of rusty sputum and subsequently began to vomit ingested food. Shortly after this he had a number of shaking chills and began to feel very warm, as though he had a "fever." He remained coherent and active, although the cough, fever and vomiting were unchanged. Three days prior to admission it was noted that he was less responsive and that he used his right arm and leg with considerable difficulty. Be became incontinent of urine and feces. A diagnosis of lobar pneumonia was made and he was started on intramuscularly administered penicillin. Shortly thereafter the coughing and vomiting subsided. However, the patient became progressively more drowsy, and twelve hours before admission he become totally unresponsive. It was noted that his respirations were rapid and accompanied by many crackles and bubbles.

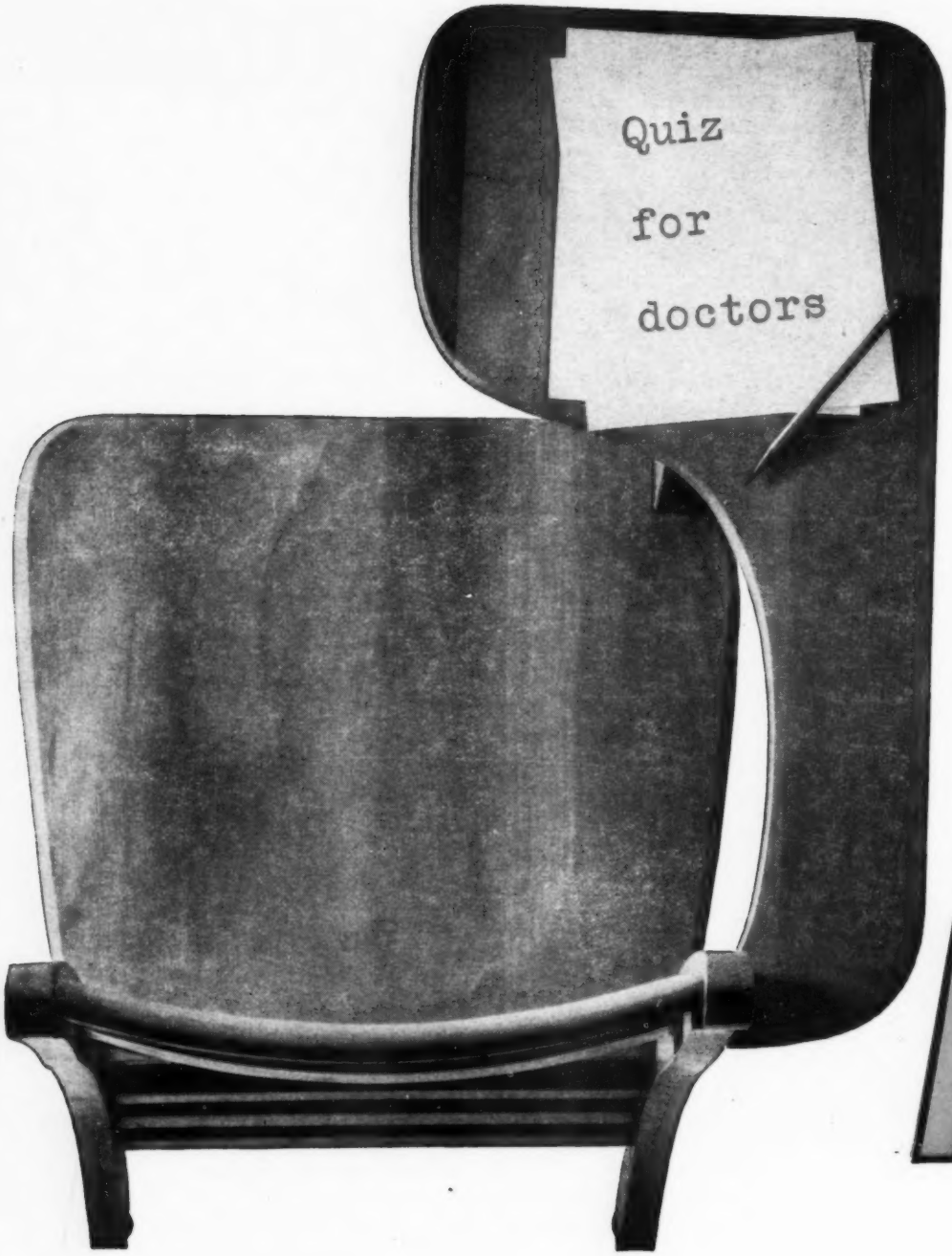
For many years the patient had "bronchial trouble," with a mild cough. One year prior to entry he had bronchopneumonia with an apparently uneventful recovery. For many years he had consumed large quantities of alcohol daily.

Physical examination revealed a sallow, semicomatose man propped up on pillows, with rapid bubbling respirations. The neck veins were flat. The heart was negative. There were coarse inspiratory and expiratory rales and rhonchi throughout both lung fields but no definite dullness. There was moderate clubbing of the fingers. There was no edema or cyanosis. He responded to painful stimuli only on the left side. The eyes were roving, and he seemed unable to see to the right. The pupils did not react. There was right-facial weakness and flaccid paralysis of the right arm and leg. The patellar, Achilles and radial reflexes were equal and active. The triceps and biceps reflexes were slightly more active on the left. Plantar reflexes were flexor.

The rectal temperature was 103.6°F., the pulse 120 and the respirations 40. The blood pressure was 150 systolic, 90 diastolic.

Urinalysis was negative except for a x albumin. A urine culture and smear were negative. Examination of the blood showed a white-cell count of 14,800, with 84 per cent neutrophils and a hemoglobin of 12 gm. A sputum smear revealed gram-positive cocci and gram-negative cocci, diplococci and rods. A throat culture showed abundant growth of alpha-hemolytic streptococci and moderate growth of catarrhalis. On lumbar puncture the initial pressure of the spinal fluid was equivalent to 220 mm. of water and the final pressure equivalent to 160 mm. of water. The fluid was cloudy yellow, and contained 7500 white cells per cubic mm., with 92 per cent neutrophils. The total protein was 1040 mg. per 100 cc., the chlorides 102 milliequiv. per liter and the sugar 37 mg. per 100 cc. The colloidal gold curve was 5555555555 and the spinalfluid Hinton test negative. Cultures of the spinal fluid grew Staphylococcus albus and B. subtilis. An x-ray film of the chest showed a clear right lung field and considerable density in the left upper lobe and probably in the lower lobe as well. A round shadow approximately 4 by 3 cm. was present at the level of the hilus. The heart was not enlarged.

The patient was started on streptomycin, penicillin and digitalis. Examination on the second day showed the following additional findings. The neck was stiff; there was pain on movement and a positive Kernig sign. The pupils now reacted to light. Eye movement to the left was normal, but to the right there was not only impairment of gaze but there was also a lack of conjugate movement, with the adducting eye not moving as far to the right as the abducting eye. The right plantar reflex was extensor and the



ACH

(you probably know every answer!)

Q. Which is today's most widely prescribed broad-spectrum antibiotic?

A. ACHROMYCIN — it's first by many thousands of prescriptions.

Q. What are some of the advantages of ACHROMYCIN?

A. Wide spectrum of effectiveness.
Rapid diffusion and penetration.
Negligible side effects.

Q. Exactly how broad is the spectrum of ACHROMYCIN?

A. It has proved effective against a wide variety of infections, caused by Gram-positive and Gram-negative bacteria, rickettsia, and certain viruses and protozoa.

Q. In what way are ACHROMYCIN Capsules advantageous?

A. For rapid and complete absorption they are dry-filled, sealed capsules (a Lederle exclusive!) No oils, no paste...tamperproof.

Q. Who makes ACHROMYCIN?

A. It is produced — every gram — under rigid quality control in Lederle's own laboratories and is available only under the Lederle label.

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left flexor. On lumbar puncture the spinal fluid was under a pressure equivalent to 230 mm. of water and contained 3500 white cells per cubic mm. The total protein of the fluid was 220 mg. per 100 cc., the chloride 118 milliequiv. per liter and the sugar 41 mg. per 100 cc., Culture of the fluid showed no growth. On the third day the spinal fluid was turbid and contained 1900 white cells per cubic mm., with 90 per cent neutrophils. By the sixth day the spinal fluid was clear and subsequently never contained more than 150 white cells per cubic mm. Physical findings remained unchanged, with the temperature ranging from 100 to 103°F. On the eleventh day the temperature rose to 105°F., the coma deepened and patient died quietly.

DR. JOHN A. EISENBEISS

The diagnostic problem today is that of a primary pulmonary disease with a cerebral complication in a 47 year old alcoholic male. The history and findings are those of chronic pulmonary disease (the clubbing of fingers) with an acute exacerbation of pulmonary infection. I will assume that the patient had bronchiectasis with a pulmonary abscess, pneumonia, and spread of infection through the central nervous system.

A few comments in regard to incidence of central nervous system complications of pulmonary disease: Of 40,000 autopsies done at the Los Angeles County General Hospital, 135 had metastatic brain abscesses; 65 were incident to disease of the lungs or pleura; 20 endocarditis; 7 septicemia; 5 liver and gall bladder infections; 3 subdiaphragmatic abscesses; 3 urinary tract infection; 3 infected spina bifida; 2 pelvic infections; 2 accessory nasal sinuses 2 tonsils; 1 each of teeth, face, esophagus and appendix; 1 incident to peritonitis; 17 were of unknown primary origin. 70 of these cases were single metastatic abscesses; 65 of the 135 were multiple.

With respect to location, the most common location was occipital and frontal, next parietal, (3) rarely, temporal, cerebellar, or brain stem, in location. In another series of 127 cases of cerebral abscess 558 were incident to bronchiestasis; 35 to empyema; 11 to purulent bronchitis; and 5 lung abscess. The age incidence of some 446 cases: 0-11 age years, 11%; 11-20 age years, 26.5%; 21-30 age years, 26.5%; 21-30 age years, 26.5%; 31-40 age years, 16.6%; 41-50 age years, 10.5%. The incidence as to sex in 576 cases: male, 389; female, 189.

Bacteriology and abcesses: The most common organisms are streptococcal, pneumococcal and stapyhlococcal, in that order. Therefore, if the impression of bronchiestasis and lung abscess is correct, the sex and age and location of this patient's brain abscess fit into the quoted statistics.

Neurologically, the patient had findings of a temporal isthmus lesion involving I assume, his major side, in that the patient was very likely right-handed, although this information is not given in the chart. The temporal isthmus is an area of white matter bounded by the vestibule of the lateral ventricle, laterally and anteriorly by the insula and posterior third of the temporal lobe. Through it run the fibers of the left occipital lobe for visual interpretation, as well as the fibers connecting Broca's area with the temporal and parietal lobes. Anterior and medial to it is the internal capsule. The patient's symptoms of right hemiparesis, involving the face, arm and leg, a right hemianopis and a right hemi-hypesthesia, as well as aphasia, though the depth of coma in the patient precluded evaluation of this, are classical for a lesion in this area. These findings are those of a subcortical lesion, and our concern is the mechanism of its development.

The acute phase of the illness, from onset to death, covers 18 days. On the day of onset the flare-up of his chronic pulmonary disease occurred. 4 days later the patient developed a bacteremia and an infected embolus lodged in the left cerebral hemisphere, or in a pial vessel over the left cortex. An area of cerebral softening developed, either an infarct or an area of red softening, due to arterial or venous occlusion then occurred. (No convulsion is noted in the history. This is frequent with subdural abscess, and may occur with subcortical abscess.) On the 7th day of his illness, an abscess is now developing in the area of cerebral softening with adjacent meningeal reaction, he became unresponsive and the right hemiparesis became a flaccid paralysis. No note of eye ground examination is made at this time. It would have been of some value to examine the eye grounds. The blood count is that of an infectious process. The findings on spinal tap, with a positive culture of staphylococcus, which is not the commonest organism found in metastatic infections from the lung to the brain; the cell, protein, sugar and chloride findings are all those of meningitis. The fact that the sputum revealed strep and other respiratory

infections, and the spinal fluid staphlococcus and B. subtilis makes one wonder if the latter organism is a contaminate, and this is of import in the differentiation of subdural abscess from subcortical abscess, in that generally, with a subdural abscess, no organisms are found in the spinal fluid.

On the 2nd day of admission, the 9th day of his illness, after administartion of streptomycin and penicillin, the patient was improved in regard to his depth of coma. His neck was now stiff, probably now noticable in that the patient's coma was less deep, and an early right sixth nerve palsy, or a loss of conjugate movement, laterally, which can occur with temporal lobe lesions, occurred; or, as described in the history, tre adducting eye not moving as far to the right as the abducting eye, suggesting that either loss of conjugate movement, or disturbance of conjugate movement, or a sixth nerve palsy was developing.

White cells in the cerebral spinal fluid were fewer in number and by the 6th hospital day, the 13th day of illness, the cell count in the cerebral spinal fluid was 150. At this time the patient should have had X-rays of the skull which would have shown a shift of the pineal body to the right. He should have then had Arteriographic studies, followed by trepination. The Arteriographic studies would have shown elevation of the sylvian group of vessels with lateral displacement and a medial displacement of the anterior cerebral group, and a subcortical abscess with cerebral softening in the region of the temporal isthmus (temporal parietal area of the brain), would have been found on trephination and needling of the area; or a depression and medial displacement of the sylvian group of vessels and a subdural abscess in the temporal parietal area would have been encountered.

Compare the course of the patient's illness with the life history of cerebral abscess; this is as follows: The infection reaches the pia or subcortical tissues by way of an entering artery, emergent vein, or a pervascular space, and because of the more abundant cortical blood supply, the infection does not localize in the cortex, but into the white matter, either by infarct (that is arterial occlusion) or hemorrhagic softening by venous occlusion. Now symptoms of encephalitis are noted. In 3 to 5 days equilibrium develops between the infecion and the reaction of the surrounding brain. The blood vessels throw off an exudate of leukocytes (the first line of defense). The blood vessels increase in size and number in the area and proliferation of connective tissue cells occur and a capsule is formed. Now the stage of battle is set. The capsule (fortification) and the leukocytes (defending troups) are pitted against the bacteria (the invaders).

On the 13th day of the patient's illness this was the local picture. During the subsequent 5 days the battle waged fiercely between the invaders and the fortifications and defending troops. The reserves, a trephination and a brain cannula to be inserted in the area, were not called into the battle, and on the 18th day the pressure and growth of organisms overwhelmed the cerebrum, and a midbrain hemorrhage, or rupture of the abscess and a subsequent pyoventricle occurred, this resulting in death.

This sequence of events could better have occurred before present-day antibiotics. The antibiotics now alter the course of the development of the brain abscess and its history is varied. Freqeuntly, only softening of the cerebrum occurs and death could have well occurred as a result of softening and edema of the involved area. There were several other factors to be considered. The patient was alcoholic. There could well have been a complicating subdural hematoma, unrelated to pulmonary disease and secondary miningitis, the patient recovering from his pulmonary disease and meningitis, only to expire incident to his subdural. The hemianopia speaks against subdural hematoma or subdural abscess, although it can be found in a cortical lesion such as these are.

My final diagnosis is bronchiectasis; pulmonary abscess; meningitis, purulent, healed; abscess, brain, left temporal isthmus, with adjacent cerebral softening and rupture into the ventricle (pyoventricle), or with a complicating pressure cone and mid-brain hemorrhage.

DIFFERENTIAL DIAGNOSIS

Dr. Maurice Fremont-Smith; A history of a patient admitted in stupor and whose family cannot be present is always unsatisfactory. Had this man had any sinus or ear infection? Had any headache preceded this condition? Because he had clubbed fingers, I should like to know how much cough he had and whether he had sputum.

Dr. Benjamin Castleman; Dr. Adams, can you answer any of those questions?

Dr. Raymond D. Adams; No.

Dr. Fremont-Smith; The urinalysis was negative except for a X albumin. I should like to know if there were any red cells.

Dr. Castleman; The urine did not contain red cells.

Dr. Fremont-Smith; The sputum smear was nonspecific. The throat culture showed alpha-hemolytic streptococcus, which is so common in the throat that it may mean nothing. The picture of the spinal fluid is interesting; it definitely shows a very severe bacterial meningitis. The chloride and sugar were low, both going with bacterial meningitis. The colloidal gold curve could have been of any type with the amount of protein that was present in that fluid. The cultures grew Staphlococcus albus, and there is no question about the fact that he had meningitis.

There was a statement that the heart was negative. I dislike statements like that. I should like to know more about the heart. The patient was given streptomycin, penicillin and digitalis. The neck veins were not engorged and the physicians thought he had no heart failure. Why digitalis? The second neurologic examination is the one that I think was the more careful one. The neck was stiff, with a positive Kernig sign. The eye movements to the left were normal, but to the right there was loss of conjugate deviation. Also on the right side there was paralysis of face, arm and leg and a positive Babinski sign, showing that he had some serious intramuscular lesion on the left. The conjugate movement of the eyes would be affected by a lesion somewhat anterior to the motor area. It is obvious that both the left motor and left sensory areas were involved, as he had lack of sensation on the same side as the paralysis. Hence, he must have had a severe lesion in the left cortex in addition to the meningitis. The interesting thing is that as the man grew worse and died the spinal fluid became better and cleared.

May I see the x-ray films?

Dr. James J. McCort; The x-ray films of the chest are not very helpful. They were taken with portable apparatus on a semicomatose patient lying in bed, and only anteroposterior films could be obtained. The right lung is clear and there is no fluid in the right pleural cavity. On the left side there is a hazy increase in density in the left upper lobe and the lobe is reduced in size, particularly in the region of the inferior and superior lingular segments. Adjacent to the hilum and in the region of the left-upper-lobe bronchus and the lingular bronchus there is an irregular soft-tissue density. A small amount of fluid is seen in the left pleural cavity. Both leaves of the diaphragm are in their normal position. No gross abnormality of the heart and vessels is seen.

Dr. Fremont-Smith; The things we know about this man are that he had a bacterial meningitis, an intracranial lesion and a condition that was called a pneumonia; it may have been pneumonia and may have cleared under the antibiotics. Apparently the antibiotics also cleared the spinal fluid. It seems to me that a collection of pus in the region of the left cortex or below the left cortex has to be considered as the focal point of the meningitis and the question is; Where did this pus arise from? It is very like a lesion below the diaphragm — a subdiaphragmatic abscess or a liver abscess that causes sterile fluid or even at times a slightly infected fluid above the diaphragm. I think the patient had something within the brain that broke through, causing meningitis, but that the meningitis was a secondary affair.

What could be the source of an infected bacterial lesion in the substance of the brain? Well, it may have been an embolic phenomenon. Subacute bacterial endocarditis, I think, rarely causes an actual abscess in the brain. An acute endocarditis with metastases might cause an abscess in the brain. That is one possibility, but there are a great many things against it. The heart was said to be negative. Almost always there is a loud systolic murmur with acute endocarditis. There was no evidence of embolic phenomena elsewhere in the body — the urine contained no red cells; the spleen was not said to be enlarged; there were no petechiae in the fingers or conjunctivas. As none of these were present, I think endocarditis is probably to be ruled out.

Another condition, — a very interesting one, — is subdural empyema — a condition described by Kubik and Adams in 1943. Subdural empyema is a condition that is not usually recognized, but if it is recognized the patient can sometimes be cured, as were two of their patients. It usually arises as a result of severe infection in the paranasal sinuses or in the ear. Dr. Kubik and Dr. Adams reported 14 cases, 13 of which arose from the sinus or the ear. There was one case that was metastatic from bronchi-

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ectasis. The symptoms are headache, stiff neck, fever, confusion, later coma, very frequently paresis or paralysis of the opposite side, a conjugate deviation of the eyes, – inability to move the eyes to the paralyzed side, – and often convulsions. In none of these cases was there an actual bacterial meningitis. There were signs of meningismus, but the cultures were negative in all cases. In no case was the cell count as high as in this case. However, the condition does need to be considered, as one can let out the pus through a burr hole, sometimes saving the patient by this procedure. I refer you to Kubik and Adams' interesting report.

The other possibility, – and the one that I thing is the most likely, – is that this man had a lesion in the chest, possibly bronchiectasis but more probably abscess. Bronchiectasis is usually bilateral, and it is often not visible in the plain x-ray films. I think he had a metastatic abscess from the lung to the brain, – either multiple or a single brain abscess, – and that such an abscess broke through the cortex and infected the meninges and that the patient therefore developed a bacterial meningitis.

Dr. Alfred Kranes; This man had clubbing of the fingers, so perhaps the chest lesion had been present for a long time. If it was, then perhaps it would be less likely to be an abscess than bronchiectasis with pneumonia–repeated pneumonias on top of the bronchiectasis.

Dr. Fremont-Smith; I think that is a good point. Repeated pneumonias are more likely to be subsequent to bronchiectasis than to an abscess.

Dr. Thomas Paine; There is one point about the bacteriologic studies; The spinal fluid grew out Staphylococcus albus and B subtilis. I think the presence of B. subtilis raises a rather large question about the nature of the real infecting organism. I do not think one can believe that Staphylococcus albus was the real infecting organism here, since the other organism was present.

Dr. Fremont-Smith; Isn't B. subtilis much more likely to be a contaminant?

Dr. Paine; Yes, therefore, I must suspect that the staphylococcus may also have been a contaminant.

Dr. John T. Stanbury; I wish the radiologist would consider the lesion in the hilum consistent with carcinoma in the lung. This might be an abscess secondary to carcinoma of the lung.

Dr. McCort; I do not see how one can exclude the possibility of carcinoma of the left-upper-lobe bronchus on the basis of this limited x-ray examination.

Dr. Stanbury; This man was an alcoholic. Such patients are quite prone to infection with Friedlander's bacillus; that can give rise to lung abscess. I wonder if that type of abscess metastasizes to the brain.

Dr. Castleman; Any type of lung infection may metastasize to the brain.

Dr. McCort; Was a cytologic examination of the sputum done?

Dr. Castleman; I do not believe one was done.

Dr. Paine; What did the gram stains of the spinal fluid show?

A Physician; We saw no organisms.

Dr. Fremont-Smith; The fact that the chloride and sugar were lowered must mean that there were bacteria in the spinal fluid.

Dr. James B. Ayer; If the time relations given here are correct, this man was well until only a week before admission. Therefore, I hardly think he had a subdural infection of the extent that is suggested, and I also think the sinuses would have shown some disease. The fact that he was well and then acutely ill suggests bacterial meningitis, perhaps with the infection more severe on one side than on the other, which is not uncommon. I have seen cases of Staphylococcus albus meningitis that ran a mild course without specific treatment. B. subtilis is a fairly common contaminant. A small abscess as an initial point of infection is possible, but I think that a thrombophlebitis is more so. Death would not necessarily be due to meningitis. I suspect that there was something else, such as a block of the cerebral fluid, perhaps from medullary pressure.

Dr. Castleman; We have the two authors of the paper on subdural empyema right here. Dr. Kubik, have you any comment?

Dr. Charles S. Kubik; Of course, metastatic subdural empyema is a rare condition. The cell count in the fluid was rather high for that, as Dr. Fremont-Smith pointed out. The rapid course might suggest that possibility, but I should think that an intracerebral abscess would be more likely.

Dr. Adams; We saw this patient on the ward. The arguments that the Medical Service used were something as follows; This man clearly had a meningitis because of all these cells in the

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spinal fluid and the low sugar. However, the lateralizing signs came on so early and were so prominent that it was obviously more than an ordinary bacterial meningitis. Usually lateralizing signs in bacterial meningitis develop rather late in the second or third week of the infection and are often due to thrombophlebitis. In this case they were the first indications of intracranial disease. We assumed, therefore, that the patient had an abscess in the brain that had ruptured into the ventricle, or a septic embolus that produced focal areas of destruction and a bacterial meningitis. Unfortunately, the history was not sufficiently complete to determine by the rate of onset which of these two conditions had occurred. There was no source of embolus. Abscess was thought most likely, but since the patient was moribund when see the Neurosurgical Service thought he would not survive a brain operation.

CLINICAL DIAGNOSIS

Metastatic brain abscess.

DR. FREMONT-SMITH'S DIAGNOSIS

Metastatic brain abscess from bronchiectasis of the lung, with rupture into meninges.
Meningitis.

ANATOMICAL DIAGNOSES

Brain abscess, left parietal lobe (bacteroides), metastatic from lung abscess.
Ventricular empyema.
Cerebellar pressure cone.
Cirrhosis of liver, alcoholic type.

PATHOLOGICAL DISCUSSION

Dr. Castleman; The autopsy showed that the pulmonary lesion was in the inferior posterior portion of the left upper lobe. It proved to be a benign abscess that apparently had produced a metastatic abscess to the brain, which Dr. Adams will tell us about. There was a fairly severe alcoholic cirrhosis.

Dr. Adams; From the time the brain was first exposed, on removal of the calvarium, it was clear that it was abnormal. The surfaces were flattened, and the left cerebral hemisphere was much broader than the right – especially its posterior half. Over the left parietal lobe the convolutions were unusually broad and flat. The arachnoidal membrane along the base of the brain and over the posterior aspect of the spinal cord was opaque and whitish, but no subarachnoid exudate could be seen on gross inspection. There was a sizable left-cerebral pressure cone, compressing the medulla. Coronal sections of the brain disclosed a large abscess occupying the left parietal lobe; it extended almost from the superior frontal region back to the occipital region and inferiorly to the temporal lobe. It was filled with pale-greenish exudate of fetid odor. When it was removed one could see that wall of the abscess was thin, rather pinkish and evidently lacking in fibrous connective tissue. The abscess extended from the cerebral cortex to the ventricle into which it had ruptured. The abscess was in a position to produce a hemiplegia, homonynous hemianopia and aphasic disorder such as were observed in this patient. The rupture into the left lateral ventricle was obviously responsible for the meningitis. Death very likely was related to the ventricular empyema and the cerebellar pressure cone.

Dr. Kranes; Was any organism cultured?

Dr. Castleman; The bacteriologic aspects were of particular interest. The abscess of the lung showed a predominance of Staphylococcus aureus, as well as many other organisms. From the brain abscess we obtained a pure culture of bacteroides. I think this is very significant and may explain the lack of response to antibiotics. Apparently the bacteroides organisms are quite unresponsive to antibiotics. Dr. Ropes, you have been studying this. Would you agree?

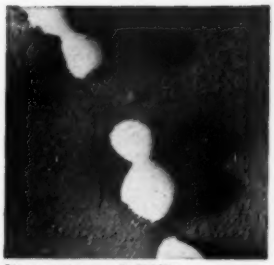
Dr. Marian Ropes; I agree; I think the evidence is still very poor concerning whether any chemotherapy or any of the antibiotics help. There are a few reported cases in which it seems possible they do. Because of the presence of Staphylococcus albus in some cultures it may have been assumed that it was the responsible organism. The bacteroides might not grow out for two weeks, and unless the cultures were kept a long while, – up to two weeks, – it might explain the absence of bacteroides in the spinal fluid.

Dr. Castleman; The staphylococci may have overgrown the bacteroides that was there.

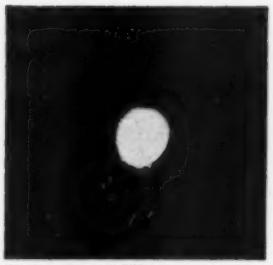
Dr. Paine; That is certainly a good suggestion. Bacteroides is a very strict anaerobe. It does not grow unless one incubates the culture under strict anaerobic conditions for a long while.

The organisms commonly involved in

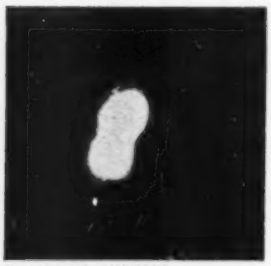
Acute Pharyngitis



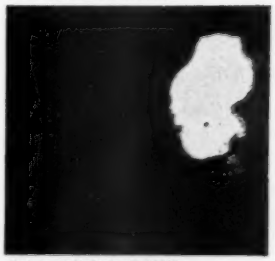
Strep. pyogenes (8,500X)



Staph. aureus (9,000X)



D. pneumoniae (10,000X)



N. intracellularis (5,000X)

H. influenzae (16,000X)

C. diphtheriae (6,000X)

K. pneumoniae (13,000X)

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THE *President's* PAGE

"AGAIN quoting from the Economic Position of Medical Care, 1929-1953, by Frank G. Dickinson, Ph.D., and James Raymond, M.A., from the Bureau of Medical Economic Research, The American Medical Association, are more facts and summary."

COMPARATIVE EXPENDITURES, 1954

Medical care expenditures for 1954 were $10,594 millions. The sum was 4.48% of the $236,532 million for personal consumer expenditures. The number of dollars spent for each of the five main breakdowns of medical care expenditures was larger than in 1953. This is also true of expenditures for recreation, personal care, and jewelry, but there were actually fewer dollars spent for alcoholic beverages and tobacco. For only two of the Department of Commerce's twelve main categories were fewer dollars spent in 1954 than in 1953: about $90 million less was spent on clothing, accessories and jewelry and about $80 million less on transportation. Medical care and death expenditures for 1954 were $578 millions greater than for the preceding year, and only $3 million of this increase was for funeral and burial expenses.

More revealing than a mere review of the dollar figures is a consideration of each of these items as a proportion of total personal consumer expenditures. Expressing each subtotal as a percentage of the whole enables the reader to see quickly how the whole was divided and that changes in spending patterns are indicated by these division. In 1954, 32.2% went for food, 25.6% for housing (excluding capital purchases) and household operation, about 10.4% for the clothing category, and about 11.4% for transportation; slightly less than 5.0% went for medical care and death expenses, while somewhat more than 5.0% went for recreation. The alterations in the spending pattern were: about 0.3% less for food, about 0.5% more for housing, 0.3% less for clothing (a continuation of a fairly definite trend since 1945) and also for transportation, a very slight increase in the proportion spent on personal care, about 0.2% more spent on personal business, and 0.1% more on medical care and death expenses. Practically all of the latter increase was for medical care and drugs.

Harry E. Thompson, M.D.
President, Arizona Medical Association.

Editorial

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The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Submit manuscript typewritten and double-spaced.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.

The Editor is always ready, willing, and happy to help in any way possible.

DR. SIPPY

THIRTY years have passed since the death of Dr. Sippy. Those who are interested in peptic ulcer would do well to review the chapter written by Dr. Sippy in the 1924 issue of Oxford Loose-Leaf Medicine in the volume on Diseases of the Digestive Tract. The first impression gained from such a review would be the thoroughness with which the subject is covered. The second impression would be the very little information which has been accumulated, or added to our knowledge of the disease during this lapse of time. Dr. Sippy was a chemist and a keen research observer as well as a great clinician. The result was that his firm convictions on the subject of peptic ulcer were proved in the laboratory as well as in the living patient. In his heyday, Dr. Sippy had a hospital service averaging two hundred patients, about 25 per cent of whom had peptic ulcers.

Today the psychiatrists, who comprise a large segment of our most brilliant minds in medicine, are carrying the ball for the responsibility of peptic ulcer. This is a very pleasant situation for the rest of us, and I am sure few begrudge them for their courage in accepting this liability. However, Koch's postulates have not been fulfilled. So we are still searching for the exact etiology of ulcer. While Sippy admitted there were a number of possibilities, he considered the bacterial origin to be by far the most probable. He reasoned that bacteria reached the end arteries in the stomach wall. Then when the nutrition to these areas in the mucus membrane was interfered with by the obstruction of these end arteries, and these areas were subjected to the corrosive action of the gastric juices, ulcer was the natural result.

There were those investigators in Sippy's time who believed that free hydrochloric acid in the stomach had no connection with the development, or presence of ulcer. One of the best known was Professor Arthur Carlson, Professor of Chemistry at the University of Chicago. Professor Carlson is still alive. Dr. Sippy's classical quotation: "Whatever the future may reveal as to the causes of ulcer and the influences that retard its healing – results – may be obtained by maintaining an efficient neutralization of free hydrochloric acid–," has withstood the test of time. Dr. Sippy demonstrated in his own chemical laboratory that the three chemical agents which produced this condition in the greatest number of cases were; calcium carbonate, sodium bicarbonate and magnesium oxide. If Sippy could return long enough to survey the present status of medical treatment of peptic ulcer he, no doubt, would be quite pleased to learn that the introduction of the so-called cholinergic blocking agents have been a tremendous aid in controlling the excessive gastric secretions. The judicious use of these drugs, along with the alkalis, constitutes the main contribution to the treatment of peptic ulcer during these 30 years.

The subject most frequently argued by clinicians during Sippy's time was the relative results of medical as compared to surgical treatment. There were those who advised routine gastroenterostomy for a time. Sippy recognized the beneficial influence of gastroenterostomy in bringing about a cure of gastric and duodenal ulcer through reduction in the acidity, and particularly through accelerated emptying of the stomach, thus reducing the duration of the corrosive action of the gastric juice. He demonstrated that the real cures, or the wonderful relief of symptoms were directly proportional to the amount of pyloric obstruction produced by the actual amount of scar tissue narrowing of the pyloric ring. Dr. Will Mayo once remarked that if he had an ulcer of the stomach he wanted to be treated nine times medically before he would submit to surgery. Sippy fully approved of gastric resection for peptic ulcer, but during his time the mortality rate in gastric resection in the most expert hands was 14 per cent. If he could return today he would be very gratified to know of the tremendous advances surgery has made in the reduction of the mortality to where it is practically nil. However, he would still bemoan the fact that we are still waiting for some one to describe the exact pathological process which occurs in producing this disease. F.J.M.

UROLOGY AWARD

"UROLOGY AWARD – The American Urological Association offers an annual award of $1000 (First prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Statler Hotel, Boston, Massachusetts, May 28-31, 1956.

For full particulars write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before December 1, 1955.

NEW FILMS

A revised list of films available through the AMA motion picture library has been prepared and copies are available upon request from the Committee on Motion Pictures and Medical Television of the American Medical Association. This catalog lists 83 medical films suitable for showing to medical societies, hospital staff meetings and other scientific groups. The catalog also includes 36 health films of interest to physicians who may be called upon to speak before lay audiences such as service organizations, Parent-Teachers' Associations, etc.

TOPICS OF *Current Medical* INTEREST

RX., DX., AND. DRS.

By Guillermo Osler, M.D.

IF you are spending ten minutes reading this column you may charge it to the big fat total of time which the average physician gives to MEDICAL EDUCATION. The Council on Medical Education and Hospitals, thru D. D. Vollan, says that the work week is 60 hours, and the time spent in 'keeping up' is 10 hours. This includes staff meetings, medical societies, formal courses, deep reading, and THIS (which should count double because people sometimes have to read it twice.)

What does the law say about 'EXPERIMENTAL THERAPY? 'Can we use methods or drugs before they do in That Other Clinic? Should we let the patient heckle us into trying a new treatment? . . . Medicine is progressive, changing, but the law demands on precedent and is conservative. 'Newness' of a method is not the same as 'experimental'; if it has been used successfully it is not experimental. Trail-blazers, however, are often near the courtroom door. They must use extra skill and diligence. They may well use the following steps, suggested by Swetlow in 'Medical Economics',—

1. Get the patient's consent in writing.

2. Be sure that the document explains the experimental nature, the risks involved. Be sure he understands.

3. Obtain corroboration from other qualified physicians of the advisibility of the procedure.

4. Have 1 or 2 qualified M.D.'s witness the job to attest to skill and diligence.

And be sure, we say, to insure.

A haunting question was also asked in 'Med. Econ.' many long months ago by Rutherford, — Do Patients ACCEPT Your Advice?" . . . A doctor, he says, most often offends his patients by giving the right advice the wrong way. Advice-giving is ticklish business, and the advice may be what the patient doesn't want to hear. . . . Some doctors have developed techniques which are nearly foolproof, which is what we all want to hear, — 1. Lead your patient to the psychological point at which he'll be most receptive to the advice. (Explain the situation. Talk.) 2. Let your patient feel he has a hand in making the decisions that will affect him. (Suggest, not dictate, advice. Let the patient talk). 3. Spare your patient embarrassment by taking a factual, dignified approach to intimate subjects. (Don't create an earthly or pool-room atmosphere). 4. Try to make your disagreeable advice palatable. (Sugar-coat the pill. Make him feel noble and unselfish. He can be helpful to his family by accepting. He is courageous. 5. Be prepared for the patient who, having considered your advice in its most beguiling aspects, still won't have any part of it. (Again, let him talk. Repeat his arguments. Tell him that he has the right of decision. And 'leave the back door open' for a change of mind).

The use of a SELENIUM DISULFIDE suspension (Selsun of Abbott Labr.) for seborrheic dermatitis has been somewhat cautious, due to fear of a chemical or allergic reaction . . . . Shapiro and colleagues of Galveston, Texas, now state that NO untoward reactions have been seen in 4 years of frequent usage.

A recent issue of the Pfizer 'Spectrum' (J.A. M.A.) starts off an article about the plethora of medical tests by saying "ONE REASON WILLIAM OSLER NEVER GOT VERY FAR is that he knew nothing about cephalin flocculation" . . . This fits the first commandment for good composition to perfection, — get their attention with a brief, punchy statement, and that lead-off is 'punchy' in every sense of the word. . . . The article suggests that the trend is towards diagnostic boxes (like Abram's), and blood analyzers. Maybe he never heard of the 'Hemo' machines which the naturopaths began to use about 1940 (and maybe still do).

The 'Year Book of Medicine' usually gives new information on well-known diseases, but the 1955-56 issue is notable for CONDITIONS AND METHODS WHICH ARE STILL FAIRLY OBSCURE, e.g., Iceland Disease, Fibrogenopenia, Hypoprothrombinopenia, Vectorcardiography of Infarction, and Primary Aldosteronism. . . . THE TITLE of a recent UCLA medical lecture is also intriguing, — 'The Oligemic Syndrome'. .It wouldn't be hard to connect it up with the word more commonly used for the condition, once you think hard. Shock, this is.

ANOTHER OBSCURE 'ENTITY' is called Sjorgren's syndrome, and it has been used as a basis for a report on the smallest possible series . . . It is supposed to consist of rheumotoid arthritis, plus lacrimal and salivary hypofunction, plus an occasional deficiency in sweating. This should be rare enough, and seldom recognized like Kartagener's triad and Behcet's syndrome. The report of ONE CASE says that Meticorten' was effective, confirming occasional good results with ACTH and Cortisone. . . .

For those who dislike names for syndromes what else would you suggest for the lengthy list of involvements called Sjogren's.[2]

If you have ever brightly said "Well, you can't die by just holding your breath!" it may come as a minor shock to know that V. J. Derbes and A. Kerr Jr. of Tulane have written a book on 'COUGH SYNCOPE,' and include a discussion of "VOLUNTARY DEATH BY BREATH HOLDING" . . . They believe that cough produces a definite syndrome. We intend to compare the discussion with that of the 'hyperventilation syndrome'.

Speaking of the HYPERVENTILATION SYNDROME, one can find more modern reports than those of the Mayo Clinic. The late J. M. Schimmenti of Oakland wrote on "The hyperventilating type of human female" (J. Nerv. & Ment. Dis.) . . . Anxiety is the basic cause. The deep breathing is often unconscious. The symptoms may come from a respiratory alkalosis (neuromuscular excitability, with twitching and tremors, hyperreflexia, etc.). Increased sensory perception may result in hyperesthesia of the skin, low pain threshold sensitivity to odors, noises, and tastes. Cold, clammy hands and feet are notable. Headaches are common, with vertigo and tension in the neck. Increased pulse pressure, tachycardia, and premature beats may be found. . . . Anxiety is basic to all of these signs and symptoms. There are phobias, poor marital adjustments, and the patient's troubles often spread to produce EMOTIONAL ILLNESS IN OTHER MEMBERS OF THE FAMILY. . . . The 'hyperventilation test' is diagnostic, — breathe deeply 60 times a minute for 3 minutes. Voila! All symptoms occur or are worse. All lab tests of blood, breath and urine reflect an alkalosis. . . . Treatment is difficult except in mild cases . . . . (Someday we may write an adjunct paper to this one. Look for it, — G. Osler, ARIZ. MED., July 1958: The Effect on the Physician of Hyperventilation Syndrome in Women).

Several articles on ACNE have accumulated this summer, and the title of one (by R. E. Osbourn in the D.C. Medical Annals) shows its urgency. — "ACNE VULGARIS, THE SCOURGE OF YOUTH" . . . Adolescence is a critical time of life. Half of adolescents have some degree of acne. All of it is unpleasant; some of it continues to scarring; and most of it causes mental and social torture. . . . Treat it early and actively. It is related to gonodal activity, but the original ideas about the connection are not true. The sebaceous glands are under the 'dyshormonal' influence (wrong androgen/estrogen balance), become overactive, plugged, infected, etc. . . . Eliminate constitutional disease and infection. Correct several dietary factors by excluding greasy and fatty foods, chocolate, excess sweets, and sea food (due to their iodine content). An Archives of Dermatology article by S. W. Barefoot suggests, since bromism can be prevented by nicotinamide, that the drug be used on the basis that iodides and bromides aggrevate acne . . . Wash the skin often with soap and water. Trade lotions, mostly containing sulfur, are useful. Evacuation of comedones and cysts may help. 'Sanding' should only be done by an expert, as should carbon dioxide slush. . . . Estrogens may be used at intervals in girls. Antibiotics are expensive, but prolonged use of small doses may be very effective. X-ray therapy is occasionally indicated. . . . Ultraviolet lamps, progesterone, and vitamin A are of no value. . . . Lawrence of San Francisco (author of 'Care of Your Skin') has written of the emotional problems in the acne patients in 'The Merck Report'. He is not impressed by a dietary influence on etiology or therapy. . . . Emotional disturbance may be a cause or effect, but it exists. A review of possible connections to ambivalence to parents, dependence vs. independence, inferiority feelings, preoccupation with sexuality, etc., all seem to this columnist to be coincidental. They may be worsened by the presence of acne, or it may be traumatized by an unhappy youth, but the psychosomatic relationship is by association and not by way of the nervous system.

SARCOIDOSIS has baffled a great many wise men of the past, including Pinner and Longcope. It still provokes arguments about diagnosis and etiology. . . . The article by Rogers and Netherton in the J.A.M.A. last year on the probable HEREDITARY TENDENCY should be remembered. Their pair of identical twins had sarcoidosis, and they found 2 other sets with the same experience. This might point to neoplasm rather than infection, heretofore the favorite cause.

Drs. Custer and Klassen have called attention to THE DIFFERENTIAL DIAGNOSIS OF BRONCHIOGENIC CARCINOMA and virus pneumonia. Twenty per cent of their 117 carcinoma patients were diagnosed wrong (and long) as 'virus' or 'primary' pneumonia. . . . Pneumonia which recurs is suspicious. A lesion which PARTLY clears, with a drop of fever, is suspicious. . . . Delayed resolution of pneumonia is of course possible, and certain types often do so (K. pneumonia, D. pneumoniae Type III, Strep., Staph., P. tularensis, septic emboli., TB., and the viruses). . . . They urge a high index of suspicion and thoroness of examination.

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ARIZONA *Pharmaceutical* PAGE

GENERIC OR BRAND NAME?

By Newell Stewart

IT HAS BEEN my experience as a retail pharmacist, a board of pharmacy secretary and as one closely connected with the pharmaceutical industry that we have much to consider on the question of medical care costs. The advent of the newer forms of medication, beginning in the middle 1930's, has provided a field of opportunity for the treatment of disease, unheard of in all past history. Total cost of medical care in the United States approximates fifteen billion dollars annually, a figure representing less than five per cent of our national income. This figure may be broken down further with the statement that total cost of medications account for about 10% of the overall medical costs of our people, presently amounting to about one and one-half billion dollars – less than ½ of 1 per cent of the national income.

It is not necessary for me to outline the increasing health consciousness of our expanding population which, together with the present earning power of the average citizen, has contributed in a large measure to the demands being made upon the health professions to provide continued advances in the armamentarium of medications. The progress we have made in drug discovery has been accomplished to some degree by the large amounts of money made available from ethical pharmaceutical firms for research studies and grants. Today our industry is expending in excess of one hundred million dollars annually for this purpose. The materials we are receiving from this investment have caused an explosion in drug therapy. Diseases are being conquered, life expectancy has been advanced, and the general health of our nation has improved at a rapidly accelerated rate. The manufacturers who have furnished the pioneering efforts in this advancement have marketed their products under brand or trade names in order to identify their preparations.

This progress of the past two decades has been made available to the public because of our heritage of incentive returns. This country of ours is not, has never been, our hope is, never will be given over to socialization. One of the most expensive ingredients entering into any prescription is the well recognzied and justified professional dispensing fee which covers not only the knowledge of the pharmacist and the time consumed in filling the prescription but also the intangible factors of responsibility imposed upon him by the laws of his city, state and nation.

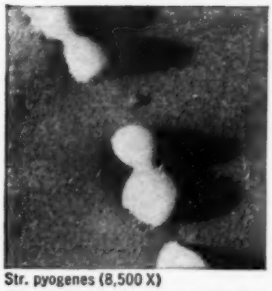
Drugs were once dispensed under the generic name system. They still are in countries where great research institutions do not exist. This system provides little incentive for "risk money" in the field of research.

Many factors enter into the acceptance of a particular brand of drug as opposed to dispensing under the generic name. These would certainly include a variable ph., disintegrating time, insoluble enteric coatings, palatability, solubility and caloric values, to name a few, which might tend to produce unanticipated results and actions for the patient.

As was quoted in the booklet "I Hate to Buy Drugs, But . . .", recently distributed by the National Pharmaceutical Council, "The public gets the benefit when an important drug really gets into mass production." When we consider the fantastic reductions made in the prices of all of our newer drugs as they have come into general use, together with the time and money-saving end results of faster recovery from so many dreaded diseases, I believe we shall have to agree that dollar for dollar we are now furnishing the public with bargain medications.

The organisms commonly involved in

Tracheobronchitis

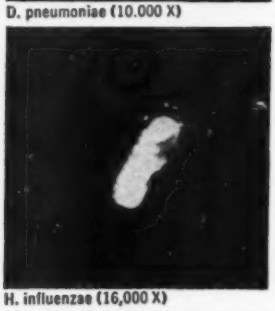


Str. pyogenes (8,500 X)

Staph. aureus (9,000 X)

D. pneumoniae (10.000 X)

K. pneumoniae (13,000 X)



H. influenzae (16,000 X)

H. pertussis (7,500 X)

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Notes from the EDITORS PEN

"MEDICAL HORIZONS" — TV PROGRAM

"Medical Horizons", a new half-hour television series, presented by CIBA Pharmaceutical Products, Inc. of Summit, New Jersey, in cooperation with the American Medical Association, will be premiered on Monday evening, September 12, over ABC-TV, as announced by T. F. Davies Haines, President of CIBA. The new documentary series will promote the American way of medical life by presenting specific accomplishments in the field of medicine brought about by the teamwork of modern medical research, education and practice.

The program will be presented each Monday evening from 9:30 to 10:00 p.m., New York time, and will be seen on a network of 45 ABC-TV affiliated stations for a minimum of 26 weeks. Stations in the Pacific and Mountain time zones will receive the program on a two-week delayed basis via kinescope film.

DOCTORS — TAKE NOTE

The House of Representatives passed HR-7225, expansion of Social Security, Monday, July 18th, by a roll call vote of 372 to 31 under a procedure (suspension of rules) barring amendments, limiting debate to forty minutes and requiring a two-thirds vote of approval. The passage of HR-7225 by the House is a sordid story. A handful of men (House Ways and Means Committee) wrote the bill in secret, denied citizens the right to express their views, demonstrating disrespect and distrust of their House colleagues. It provides for the compulsory collection of an additional $2 billion per year of taxes and compels another 200,000 individuals to participate in the unsound and immoral sceme of Social Security. The Senate Finance Committee will hold open session to hear administration views and decide whether or not to have public hearings.

The AMA opposes the section of the bill that would set up a national compulsory disability insurance program, maintaining that such a program would project the government into the medical care field because of the necessity for regulating medical determination for disability. Your Association joined AMA in vigorously opposing the House decision to deny public hearings on so important a measure and opposing enactment of the bill at this time.

Mrs. Hobby said that no action should be taken in this direction at least until HEW has had time to evaluate operations of the "waiver of premium" enacted last year. She also said Congress should learn more about the relationship between rehabilitation, for which a new program was enacted last year, and cash payments for disability, implying that the temptation of cash payments might lessen a disabled worker's interest in rehabilitation.

The Congressional Record of July 18, 1955, page 9304, shows that Representative Rhodes VOTED FOR passage of HR-7225, and that Representative Udall did not vote but was paired with Mr. Crumpacker of Indiana. This was a "general pair", therefore it is not possible to ascertain whether or not Mr. Udall favored passage. In any event none of our Representatives positively opposed passage of this bill.

BOARD OF MEDICAL EXAMINERS — STATE OF ARIZONA

Of interest to all members of the medical profession is the recent appointments of Governor McFarland to membership on the Board of Medical Examiners of the State of Arizona, July 25, 1955, for terms of three (3) years each expiring June 30, 1958. They are: Carl H. Gans, M. D. (reappointed) of Morenci, and, Zenas B. Noon, M. D. of Nogales. They with present incumbents Drs. Harry T. Southworth, President, (Prescott), Orin J. Farness, First Vice President (Tucson), and Maurice R. Richter, Secretary-Treasurer (Phoenix) comprise the Board membership of five. Dr. Gans is Second Vice President.

"RECENT INFORMATION ON ECONOMIC POISONS AVAILABLE"

New medical problems have arisen since the very great increase in use of agricultural chemicals including insecticides and pesticides. Since many of our readers are in portions of Arizona where such agricultural chemicals are used extensively in crop dusting operations and otherwise, we call attention to the latest annual revision of "Clinical Memoranda on Economic Poisons." This booklet is prepared by the U. S. Public Health Service, Communicable Disease Center, Technology Branch, Savannah, Georgia and is being made available free upon request to the National Agricultural Chemicals Association, 1145 19th Street, N.W., Washington 6, D. C. It is much more comprehensive and informative than its modern title suggests.

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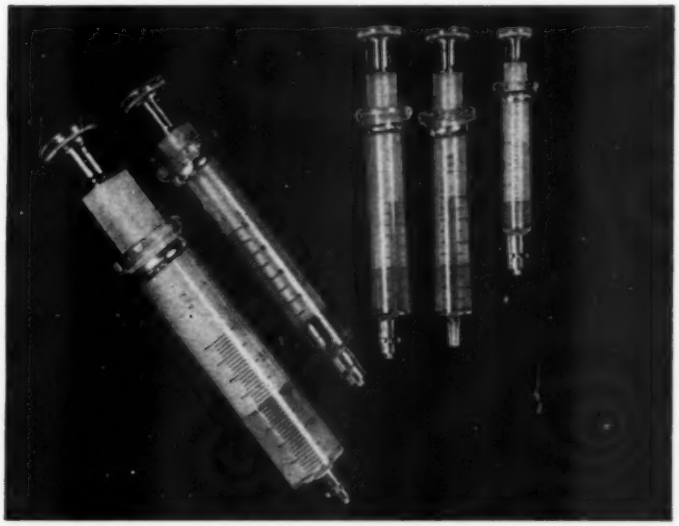

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Organization PAGE

CIVICS

Norman Ross, M.D.

PHOENIX INDIAN CENTER, 28 Wall Street, Phoenix, Arizona, Leon Grant, Director.

TO FOLLOW is a short history of the Phoenix Indian Center and a list of its hopes and needs. To our knowledge there are no other non-sectarian Indian centers in the state and only one church center is maintained and that in Flagstaff, Arizona. It is interesting to note that probably the largest and best known Indian Center is in Los Angeles, that this center was actually sectarian in origin, Quaker, and that it is now operated and financed entirely by the Indians themselves.

The Chairman of the Board, Mr. Walker, sent samples of literature produced by the Director and Board plus that of the Indian patrons. He advises that the Phoenix Indian Center would be pleased to furnish like material to physicians, to medical associations or other organizations that are attempting to improve the lot, health, and social position of the American Indian.

He states that they are particularly interested in continuing the interest of the medical profession and our help as he feels that only physicians can cut down the incidence of disease and ill health among the Indians and thus curb the spread of diseases which are so common among the Indians to the white man. He feels that the health of the Indian is of equal importance too, and would probably be the greatest single item in the process of integration.

The need for the Phoenix Indian Center was discovered by a court probation officer. It was he who pioneered the social agency, contacting the Cook Christian Training School in Phoenix because of problems of delinquency among the Indians.

The following is quoted verbatim:

"The only central location that could be had for low rent was off a dirty alley ironically named Wall Street. There, in the basement at No. 28, was a filthy room that could be rented for $10.00 a month. Above it was a liquor warehouse of sorts where during the hot weather the beer bottles would explode and the beer would seep thru the ceiling.

Dr. Nace of the Mission Board of the National Council of Churches (Lutheran, Presbyterian, Baptist, Methodist, etc.) was contacted and the Board allocated $2600 to clean up the place, hire a director of activities, etc.

Until this Center was set up, there was no place for the Indian coming in from the Reservations to go except to the undesirable places in town where he was made welcome but which were detrimental to him and his family.

Many Indians do come from the Reservations looking for work, for homes and for church homes. The aim of the Center is to help these people.

Judge Pensinger has told us that he has found much assistance from the Center in an understanding of how to deal with Indian problems. Invariably he seeks the counsel of Leon Grant in Indian cases.

As the Center grew in popularity and needed larger and better quarters, they added three rooms upstairs, bringing the rental up to $55.00.

The John F. Long Construction Company donated the material and labor to plaster and renovate the Center, installed necessary plumbing and other features.

In 1954 the National Council withdrew its financial aid and a group of Phoenix business men assumed the responsibility. They were in charge for a year and in February 1955 it was requested that the Cook Christian Training School assume the responsibility of financial and whatever other aid the Center needed.

Mr. Leon Grant, an Omaha Indian, is in charge of the Center at a salary of $250.00 a month. The Cook Christian Training School furnishes him and his family a house on its Campus. He is a graduate of this school. His wife is a Navajo and they have four children.

LOOKING AHEAD

At present the Center is open only until 5 P.M. It needs additional funds to enable it to hire an assistant director who would take over in the evenings, during vacation times of the regular director and at other times when he must be away from the Center. Evenings are times

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when bars and other questionable places attract those who have no other place to go.

It is hoped that in the Fall the Center may have some activity for each night except Sunday. These are some of the plans they have in mind.

One night a week for Indian fellowship and prayer meetings, led by people of the various churches in the Valley.

One night of movies. The Standard Oil Company will release films to the Center when they can supply a projector and screen.

One night a week for teenagers.

One night a week for Indian Arts and Crafts and telling of Indian legends.

One night a week for library.

It is also hoped that they can fix up the basement for recreational purposes such as Ping Pong, Pool, Checkers or other games. The basement will have to be replastered and painted.

APPEAL

Some people have seen only the unpleasant side of the Indian. However, is it not the fault of the White Man that the Red Man has not had the advantages to help him grow into a desirable citizen? With understanding and education the Indian can climb to unheard of heights. One old Pima saint put it this way, "My white friends, you Christians are pulling us up out of the hole of bad living. PLEASE, Mr. White Man don't let go that rope."

LIFE INSURANCE ASSOCIATION OF AMERICA, 488 Madison Avenue, New York 22, New York.

The Editor-in-Chief forwarded to this office **two pamphlets,** one the Preliminary Report on the Annual Survey, the Extent of Voluntary Insurance Coverage in the United States; the other pamphlet consisted of a number of charts sent by the Health Insurance Council of this same agency. As has been previously reported the Health Insurance Council and the **Life Insurance Association of America will furnish you** this most interesting **material request.**

In that voluntary health insurance coverage has been reported at the national level on numerous occasions on this page, and in that the Blue Cross and Blue Shield Committee's reports are regularly published and both show phenomenal growth and expansion, it would seem that some form of local review would be of interest.

It is entirely probable that **our patients do not know the amount and kind of insurance coverage that they have purchased.** It occurs to us that good medical care of our patients must include obtaining for them the benefits of the one or more prepaid plans that they own. They should expect us to be more familiar with prepaid voluntary medical care programs and policies than they are, in that we have endorsed, recommended, and actually presented a prepaid insurance plan of our own.

How can we estimate the amount of our practice for which our patients can receive compensation from that prepaid voluntary insurance policies? A week's review showed that insurance coverage for office care was to all practical purposes non-existent. Exceptions were the industrial cases and a few of our patients who were covered for laboratory procedures. For the time being it was decided to limit our study to our hospital cases.

A call to a local hospital and the business office of same produced interesting data. Advice was to the effect that of their approximately 1500 admissions per month, 50 per cent had Blue Cross coverage, 30 per cent private insurance, 5 per cent were Arizona State Industrial Cases, one per cent or less were paid by the Federal Government and less than one per cent were hospitalized by social agencies.

From the above you can see that approximately 80 per cent of persons requiring hospitalization were covered by voluntary prepaid insurance programs. Only 13 per cent of hospital admissions completely pay their own bills.

A review of the out-patient clinic or the emergency service of approximately 100 cases per month, shows that 20 to 25 per cent of these cases are State Industrial cases, and that less than 20 per cent are accidents not related to employment and paid in part by Blue Cross. Any estimate of the number of persons who have private insurance was not obtainable because in these cases the office insurance procedure is completed by the private physicians and the patient needs only a receipt for hospital care. The office did not remember any emergency cases coming under Federal or social agency care.

Our friend in the business office of the hospital who furnished this data stated that she remembered just a few years ago when their office had very few prepaid insurance cases. They had many less employees then and no

one employee assigned to "insurance". She stated that four persons now do nothing but complete detailed insurance reports for insurance companies and Blue Cross, and she estimated that 90 per cent of the remainder of the office employee's time was taken up by insurance cases and insurance business. Though the insurance practice in a private office appears small today, if our experience follows that of the hospital, we may look for its increase by leaps and bounds. A study of the national programs shows that there is to be an extension of present coverage of the private programs. The National Blue Cross is recommending expansion and extension of its program and that of its hundreds of affiliates. Medical pay is now a part of the Arizona State Blue Cross and Blue Shield program and contract medicine, some say, is now ever more advanced than other programs in our state.

We must in the immediate future see that our most cooperative patients, those who have taken our advice and arranged to prepay their hospital, surgical, and medical bills, "gets his money's worth".

AMERICAN CANCER SOCIETY, INC., ARIZONA DIVISION, 1429 North 1st Street, Phoenix, Arizona.

A new booklet entitled, "Cancer Detection in the Physician's Office", recently reviewed by the Medical and Scientific staff, and endorsed by the Professional Advisory Committee of the American Cancer Society, will currently be mailed to all practicing Physicians in Arizona through the courtesy of the Arizona Division, American Cancer Society.

Doctors will find this booklet well prepared, written in an interesting fashion and generously illustrated. The Arizona Division hopes it will prove a worthwhile addition to each Doctor's office in their fight against the nation's second greatest killer, CANCER.

WE'VE BEEN PLEDGED

J.A.M.A. Feb. 19, 1955, Page 666, Organization Section:

"The Board of Trustees of the American Medical Association issued a statement commending President Eisenhower for his Jan. 31 health message to Congress and has pledged continued support to improve the health of the nation by strengthening medical efforts at the state and local levels." ***Following is an extract of the statement released by the Board of Trustees on the recommendations of Mr. Eisenhower that are supported in principle by the Board:

"1. An 'intensified attack' on mental illness, which has been termed 'the nation's number one health problem' by the A.M.A. Council on Mental Health.****This project co-sponsored by 16 national organizations, expects to evolve some fundamental departure from traditional concepts of dealing with mental illness which will lead to a far more effective attack on the problem than has thus far been realized".

Statement by Dr. David B. Allman Before Subcommittee of House of Representatives, March 10, 1955, J.A.M.A., April 9, 1955, Page 1315, Organization Section:

Dr. Allman reports on the activities and program of the American Medical Association Committee and the Committees on Mental Health of the State and County Medical Associations. He, as representative of the American Medical Association, outlines a program and calls for the establishment of cooperative relationships of the membership of the American Medical Association with other national groups in the field of mental health.

"In conclusion, I wish to emphasize that the American Medical Association is keenly aware of the seriousness of the mental health problems which exist today and is vitally interested in doing everything within its power to insure that the medical profession makes its maximum contribution in their solution."

THE ARIZONA ASSOCIATION FOR MENTAL HEALTH, Joe M. Young, President, P.O. Box 4664, Tucson, Arizona.

A conference with the office of Dr. Robert S. Barrett, Director of the Maricopa Child Guidance Clinic, shows that on Thursday, March 10, 1955, the Arizona Association for Mental Health which had been described as dormant, stirred; dined and reorganized. The program of the Association for Mental Health is a unique one. Here a private social agency operates through governmental agencies, matching federal funds play an important part in this agency's budget.

The names of those private citizens who have assumed board and committee appointments are most impressive. This speaks well for the success of the present organization as we may anticipate such persons stimulating governmental agencies to increased activity in a search for more federal funds to match local funds.

President Eisenhower's interest is reported as

well as the pledge of the American Medical Association and its component associations.

In Maricopa County at the local level the Maricopa County Medical Auxiliary is the continuing force supporting the Maricopa County Child Guidance Clinic.

An organization of private citizens in Pima County have developed and promoted the Tucson Child Guidance Clinic. Private citizens in Yuma and Nogales are at present in the process of organizing.

From the above we can see that the success of this program has been pledged by medicine, that we have assumed individual and professional responsibility. Here is a newly organized social program of vital importance. Your participation at policy level will be welcomed.

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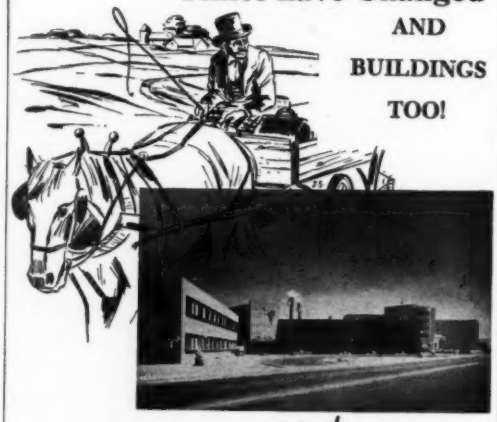

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THE BIBLIOGRAPHY OF EDWARD WILLIAM ADAMSON, M.D.

This column will contain from time to time different episodes in the history of medicine in Arizona, as reflected in the stories of it's hospitals, medical buildings, medical societies, pioneer doctors, etc. It is sponsored by the Committee on Medical History of the Arizona Medical Association. Any contributions to this subject are always welcome. — ED

"Douglas, June 11, 1955 (AP) — Dr. Edward William Adamson, 75, veteran Douglas physician, has been nominated by the Cochise County Medical Society as the outstanding Arizona physician of 1955.

"A practicing physician here for 50 years, Adamson has been superintendent of the County Hospital here since 1924."

— Phoenix Gazette, June 11, 1955.

DR. ADAMSON was born at Cass City, Michian, on September 23rd, 1879. He was the first child and the only son of James Walker Adamson (Scotch), and Emma Mertz (German). On the map of Michigan, Cass City may be found amidst the finest pine ever cut. In 1884 the Adamson family moved to Bay City, Michigan, located along the Saginaw River, a city made prosperous by 36 lumber mills.

At the age of eleven Edward Adamson, like many of the boys of Bay City, went to work during summer vacations in the lumber mills, first as a tally-boy keeping score of the lumber as it was loaded on boats for shipment; next as a tail-carriage man, then scaler behind the trimmers, apprentice in a machine shop, and one summear as chainman on a lumber surveying crew. One summer he was an oiler aboard a lake tug going to Canada for logs.

As both of his parents were musical, Dr. Adamson's musical education began early when he learned to play the cornet and clarinet. His father taught bands. At the age of 16 Edward Adamson was playing the violin publicly in dance orchestras, and occasionally as second violin in the opera house orchestra. He never lost his love for music and after he moved to Douglas he organized and conducted his own orchestra whose members played together for their own amusement for over ten years. Dr. Adamson considers this orchestra as his first "hobby".

At the age of seventeen Edward Adamson graduated from Bay City High School where he had taken a mechanical course, including physics and mathmatics in addition to courses in drawing and shopwork and the required high school curriculum. His father was a teacher of languages, but later became a pharmacist, so he insisted that Edward take four years of Latin, two of Greek, and also German. This knowledge later proved excellent preparation for his studies in medical school.

During the three years following graduation from high school, Edward Adamson worked as a machinist's helper and on a surveying crew, and at nights he worked in a pharmacy. He made the acquaintance of a doctor in Bay City while repairing some of the electrical instruments in his office. Here he was allowed to read some of the doctor's books and familiarize himself with office routine, all of which guided him in the selection of his life's work. Financial arrangements by the doctor enabled the young man to go to medical school and in 1900 he entered the University of Michigan to study medicine.

Because of Adamson's ability as a draftsman and artist, he came to the attention of Professor G. Carl Huber in the department of anatomy, who was then occupied in writing a book on Histology. Adamson made the drawings for this book in his spare hours and on Sundays. Professor Huber always "touched up" the drawings but he nevertheless gave Adamson credit for the art work. The two men became lifelong friends. Sometime later Professor Huber directed his young student in the construction of wax models of the sudoriferous glands of the skin, magnified 700 times, showing the anatomy of the sweat-glands from fetus to adult. These models are still on display at the Medical Museum in Ann Arbor, Mich.

In 1903 Professor Huber described this work in a volume dedicated to Dr. Victor V. Vaughn, Dean of the School of Medicine, and entitled "Contributions to Medical Research". Adamson also did much of the animal surgery for Professor Huber's study of nervous tissue. This same year Dr. Adamson received the honor of

Fellow of Sigma Chi. On his graduation from medical school he secured an internship in the hospital of the Calumet-Hecla Mining Company, Calumet, Michigan. This was considered one of the best assignments available. Here he obtained wide experience in all branches of practice and the opportunity to do the actual work under the immediate supervision of the regular staff members.

On the 29th day of May, 1905, Dr. Adamson arrived in Douglas, Arizona, in answer to a request by Dr. Wright who was then owner and chief of staff of the Calumet Hospital. He wished to go to Paris for a year's post-graduate study and engaged Dr. Adamson as locum tenens. The following year when Dr. Wright returned, Dr. Adamson set up his own private practice and was very successful in every way. In addition to general practice and obstetrics he did a great deal of surgery.

The year 1910 saw the completion of the New Cochise County Hospital and the appointment of Dr. Adamson as Superintendent. He was sent to New York to purchase new equipment for the entire hospital, including everything needed down to the kitchen knives. Dr. Adamson remained as superintendent of the County Hospital for two years, but the hospital was considered a political plum and for the next twelve years 16 different doctors held the position of superintendent until 1924 when the hospital was taken out of politics. Once again Dr. Adamson was asked to accept the position of superintendent and he has retained this position ever since. After much hard work and many disappointments the hospital was built into a really superior institution and compares favorably with any of similar capacity in Arizona.

While taking some post-graduate work in local anesthesia at Chicago in 1924 Dr. Adamson met an Austrian surgeon who was demonstrating spinal anesthesia in this country. He became interested in the technique and upon returning to Douglas he began to use the new method. To date, in about 6000 spinal anesthetics, he has never had any difficulty or untoward result with spinal anesthesia.

When Dr. Wright retired in 1926 he sold the Calumet Hospital to Dr. Adamson who operated it until 1931 when the Calumet and Arizona Mining Company merged with the Phelps Dodge Corporation. Once again Dr. Adamson entered private practice, limited to diagnosis and surgery, in association with Dr. Jack Hild.

In 1913 Dr. Adamson attended a meeting of the Surgeons of North America in Chicago and the following year he was elected a Fellow of the American College of Surgeons.

During the First World War Dr. Adamson was commissioned a Captain in the Medical Corps in 1917 and assigned to duty with the Army Air Force, first at Lake Charles, Louisiana, then Long Island, New York, and finally at Hampton, Va. While in the service Dr. Adamson took 30 hours of flying time, but now he considers himself too old for flying. He was promoted to the rank of Major and in March, 1919, honorably discharged from the Army and returned to his private practice in Douglas.

In social, civic, and fraternal activities Dr. Adamson has been quite active. He has been a member of the BPOE since 1900 (55 year member), and was Exalted Ruler of Douglas Lodge 955 during the year 1923. He became a Mason in 1910 and three years later was elected Master of the Higher Degrees of Masonry, Chapter Royal Arch and a 32nd Degree member in the Scottish Rite. When the Kiwanis Club of Douglas was formed Dr. Adamson was a charter member and its president in 1925. He served on the Board of Directors of the First National Bank of Douglas for six years, until the Valley National Bank acquired the First National. He is a member of the Cochise County Medical Society, the Arizona Medical Association, the American Medical Association, and the Southwestern Medical Society.

From 1912 to 1945 he was Acting Assistant Surgeon U.S. Public Health Service assigned to duty at the Douglas Port examining aliens entering this country from Mexico. For 25 years Dr. Adamson was School Doctor for the Douglas public schools, work which he enjoyed very much, particularly since he helped eradicate the serious eye disease trachoma in this locality.

He has been an ardent out of doors sportsman since his youth; adept at swimming, ice skating, semi-professional hockey, and sail-boating on the Great Lakes. He still enjoys hunting water fowl and upland birds, camping, and he has the reputation of being an excellent camp cook, attested to by all who have eaten his fare.

Among his many hobbies he excels at metal work and carpentry, particularly in the branch of inlaid wood pictures. His workshop repre-

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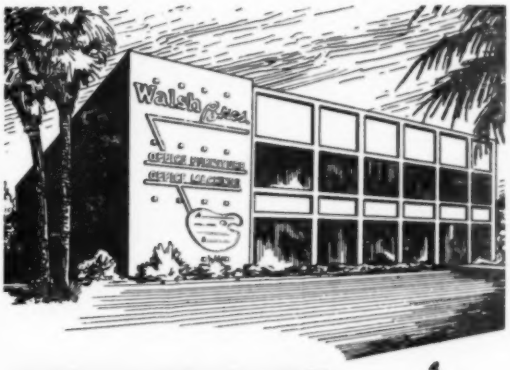

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sents an investment of about $16,000 in fine equipment, and he has several patents which return him sufficient royalty to maintain his shop. He does a great deal of photography, both "still" and "movie", which takes time and money to continue.

He purchased a Ranch-Orchard near Portal, Arizona, in 1932 where he raised apples, registered cattle and polo ponies. He sold his ranch in 1953, considering it a great educational venture but not highly successful as a financial investment.

He was married to Miss Florence Dillingham of Boston, Mass., in 1906. She died in 1924. Two years later he re-married, his second wife being Miss Anna Van Brunt Foster of New York City. She passed away in 1944, at Douglas. Dr. Adamson has no children.

Dr. Adamson believes his life has been a great adventure which has yielded many things, both happy and disturbing, many sincere friends and grateful patients, for which he is duly thankful.

Arthur E. Engelder, M.D.
Warren, Arizona

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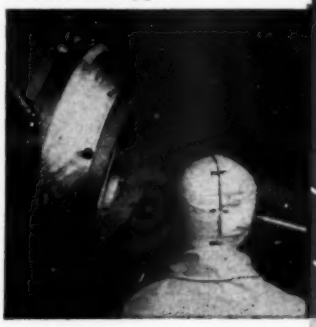

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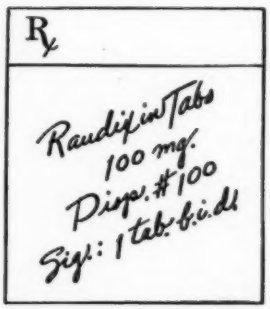

*Ataractic, from ataraxia: calmness untroubled by mental or emotional excitation. (Use of term suggested by Dr. Howard Fabing at a recent meeting of the American Psychiatric Association.)

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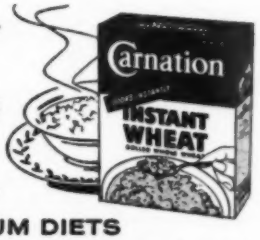

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BASIC CEREAL BREAKFAST

ITEM	WEIGHT OF SERVING gm.	PROTEIN gm.	ENERGY calories
Fruit	77	0.4	68
Cereal (dry wt.)	30	3.2	110
Enriched White Bread (toasted)	50	4.2	130
Sugar	10	0.7	40
Butter	10	0.1	73
Whole Milk	480	16.9	330

Calories	750	Fat (gm.)	28
Protein (gm.)	25	Carbohydrate	100

*Literature available upon request. Address Nutritional Research Dept., Albers Milling Co., A Division of Carnation Co. 5045 Wilshire Blvd., Los Angeles 36, Calif.

Pork in the Dietary

During Pregnancy and Lactation

CERTAIN NUTRIENTS are required in greater than normal amounts during pregnancy and lactation. Pork meat, though its cost is low, supplies a remarkably high quantity of the nutrients required by the maternal organism in these periods of physiologic need.

During pregnancy the maternal organism may store 3.3 to 5.5 pounds of protein in excess of that contributed to fetal tissue.[1] Enough iron is stored to approximate the entire amount secreted in the milk during 9 months of lactation, in addition to the iron supplied to the fetus.[2]

The body of the newborn infant contains approximately 500 grams of protein, 14 grams of phosphorus, and 0.5 gram of iron.[3] It is estimated that the lactating mother, through breast milk, provides a 26 week old infant with about 12 grams of protein, 76 grams of lactose, and 1.2 mg. of iron each day.[2]

Pork meat, an excellent source of high quality protein, thiamine, niacin, and iron,[4] also supplies valuable amounts of other B vitamins, as well as phosphorus, magnesium, and potassium. The thiamine content of pork is particularly important, since there are few more valuable food sources of this vitamin.[4]

Pork and pork sausage—economical, good tasting—are valuable components of the dietary of the pregnant or lactating woman. Just how valuable, is shown in the table below.

1. Toverud, K.U.; Stearns, G., and Macy, I.G.: Maternal Nutrition and Child Health, an Interpretative Review, Washington, D.C., National Research Council, Bull. 123, 1950.

2. McLester, J.S., and Darby, W.J.: Nutrition and Diet in Health and Disease, ed. 6, Philadelphia, W.B. Saunders Company, 1952, p. 241.

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5. Watt, B.K., and Merrill, A.L.: Composition of Foods—Raw, Processed, Prepared, Washington, D.C., United States Department of Agriculture, Agricultural Handbook No. 8, 1950.

6. Bowes, A. deP., and Church, C.F.: Food Values of Portions Commonly Used, ed. 7, Philadelphia, Anna dePlanter Bowes, 1951.

Percentages of Recommended Daily Dietary Allowances* for Pregnant (3rd Trimester) and Lactating Women Provided by 3-Ounce Portions of Cooked Pork Meats and Pork Sausage

	Protein	Iron	Phosphorus	Thiamine	Riboflavin	Niacin	Calories
PREGNANCY (3rd trimester)							
Ham, without bone, 3 oz., cooked[5]	25.0%	17.3%	13.5%	30.0%	10.0%	26.7%	12.5%
Pork Chops, without bone, 3 oz., cooked[5]	25.0%	17.3%	13.3%	47.3%	10.0%	28.7%	10.5%
Pork Sausage, 3 oz., cooked[6]	17.3%	14.0%	9.2%	27.7%	10.1%	18.5%	14.7%
LACTATION							
Ham, without bone, 3 oz., cooked[5]	20.0%	17.3%	10.1%	30.0%	8.0%	26.7%	10.2%
Pork Chops, without bone, 3 oz., cooked[5]	20.0%	17.3%	10.0%	47.3%	8.0%	28.7%	8.6%
Pork Sausage, 3 oz., cooked[6]	13.8%	14.0%	6.9%	27.7%	8.1%	18.5%	12.0%

*Recommended Dietary Allowances, Washington, D. C., National Academy of Sciences—National Research Council, Publication 302, 1953

The nutritional statements made in this advertisement have been reviewed and found consistent with current medical opinion by the Council on Foods and Nutrition of the American Medical Association.

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Optimal dosage proved to be one envelope (7 grams) of Knox Gelatine administered daily for three months. Improvement, however, was noted after the first month. If you would like more complete details of this work, just use the coupon.

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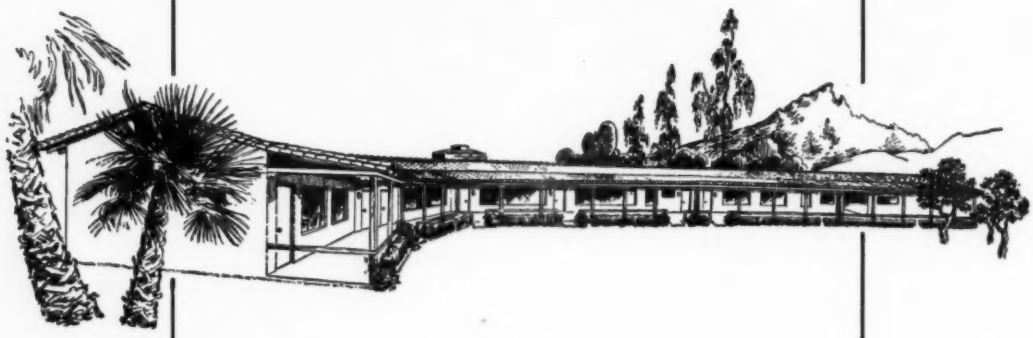

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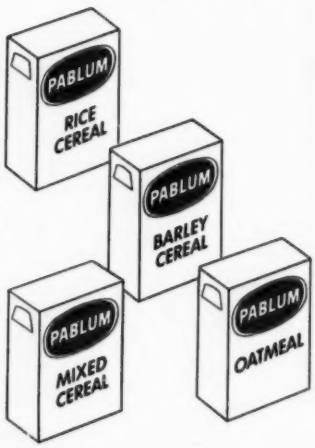

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